JOURNAL
OF THE
American Veterinary Medical Association

FORMERLY
AMERICAN VETERINARY REVIEW
Original Official Organ U. S. Vet. Med. Ass'n.

Edited and Published for the

AMERICAN VETERINARY MEDICAL ASSOCIATION
By
H. PRESTON HOSKINS, CHICAGO, ILL.

EXECUTIVE BOARD

A. E. CAMERON, 1st District; E. P. ALTHOUSE, 2nd District
L. A. MERILLAT, 3rd District; WILLIAM MOORE, 4th District
H. C. H. KERNKAMP, 5th District; I. E. NEWSOM, 6th District
W. A. SULLIVAN, 7th District; L. J. ALLEN, 8th District
H. W. JAKEMAN, *Chairman*, 9th District; F. A. ZIMMER, 10th District
CHAS. W. BOWER, Member-at-Large
OSCAR V. BRUMLEY, ex officio; H. D. BERGMAN, ex officio

Volume XCI

NEW SERIES VOLUME 44
July, 1937, to December, 1937
CHICAGO, ILL.
1937

INDEX OF AUTHORS

Allott, A. J., 588.
Amadon, R. S., 674.
Arnold, Aaron, 515.

Backus, N. D., 64.
Bishop, Lucille M., 22.
Bloom, Frank, 679.
Boyd, W. L., 22.
Bullis, K. L., 48.

Craige, A. H., Jr., 674.

Davis, C. L., 209.
DeCamp, C. E., 581.
Delaplane, J. P., 317.
Delez, A. L., 332.

Edwards, P. R., 656.
Elevehjem, C. A., 515.
Emmel, M. W., 59, 201, 205.
Erwin, L. E., 317.

Farquharson, James, 551.
Fechner, Robert, 276.
Ferguson, L. C., 163.
Fitch, C. P., 22.
Foster, Robert J., 268.
Frank, E. R., 639.
Frick, E. J., 571.

Graves, Earl F., 97, 216, 606.
Greenwood, D. A., 555.
Grinstead, Emil E., 208.
Guilbert, H. R., 193.

Hammersland, Hazel, 186.
Harshfield, G. S., 323.
Hart, G. H., 193.
Hawn, M. C., 323.
Hell, Henry, 544.
Henderson, E. W., 146.
Henley, W. W., 59.
Hewitt, E. A., 555.
Holle, H. A., 310.
Hull, F. E., 656.

Jacobs, Philip P., 714.
Joneschild, E. M., 186.
Jungherr, Erwin, 73.

Kleaveland, I. J., 647.

Lames, G., 334.
Lee, C. D., 146.

McGregor, A. N., 209.
McNutt, S. H., 532.
Madsen, D. E., 206.
Marsh, Hadleigh, 88, 330, 600.
Mericle, R. B., 651.
Mitchell, J. F., 176.
Mohler, John R., 288, 597.
Morley, L. C., 94.
Murray, Chas., 146.

Nelson, V. E., 555.

Roderick, Lee M., 323.
Rodier, E. A., 294.
Rouse, P. L., 98.

Shillinger, J. E., 94.
Stiles, George W., Jr., 209.
Stuart, H. O., 317.

Taylor, J. B., 182.
Tunnicliff, E. A., 600.

Van Roekel, H., 48.

Wantz, G. E., 571.
Wilcke, H. L., 146.

LOUISIANA STATE UNIVERSITY LIBRARY

1/8/47
Sent
from
Ag Bldg

JOURNAL
of the
American Veterinary Medical Association
Formerly AMERICAN VETERINARY REVIEW
(Original Official Organ U. S. Vet. Med. Assn.)
H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

A1.594
2

COL. R. J. FOSTER, Pres., Washington, D. C.
M. JACOB, Treas., Knoxville, Tenn.

EXECUTIVE BOARD

CASSIUS WAY, Member-at-Large and Chairman
COL. R. J. FOSTER, ex officio; O. V. BRUMLEY, ex officio
A. E. CAMERON, 1st District; E. P. ALTHOUSE, 2nd District
L. A. MERILLAT, 3rd District; WILLIAM MOORE, 4th District
H. C. H. KERNKAMP, 5th District; I. E. NEWSOM, 6th District
C. H. HAYS, 7th District; L. J. ALLEN, 8th District
H. W. JAKEMAN, 9th District; F. A. ZIMMER, 10th District

The American Veterinary Medical Association is not responsible for views or statements published in the JOURNAL, outside of its own authorized actions. Reprints should be ordered in advance. Prices will be sent upon application.

Vol. XCI, N. S. Vol. 44 JULY, 1937 No. 1

BACK TO OMAHA

Back to Omaha after 39 years. A long time between visits. Much history, scientific and otherwise, has been recorded in the interval between 1898 and 1937. Veterinary practice then was almost entirely equine. It was three years later that the first cow to be presented at an A. V. M. A. clinic was led into the arena at Atlantic City. A few veterinary entrepreneurs had been far-sighted enough to provide for the hospitalization of small animals in their recently constructed "infirmaries." Many states were without veterinary practice acts and, in quite a few, state associations had not been formed. Veterinary conferences and short courses had not yet come upon the scene. Three veterinary journals were struggling for an existence. One survived—*The American Veterinary Review*, which later (1915) became the JOURNAL of the A. V. M. A. Veterinary supply houses, as we know them today, did not exist and the profession had to depend on several well-known manufacturing pharmaceutical houses for their tinctures and fluidextracts, active principles and powdered drugs. Every veterinarian was his own pharmacist.

Serum therapy was in its swaddling clothes at the time of the first meeting in Omaha. Tuberculin was something of a scientific curiosity and bacterin therapy was as yet unborn. The x-ray had been discovered three years before but had not been put to practical use in the field of medicine. Motor cars were

619.05
V.91
304587

in operation but practitioners depended upon the horse and buggy for getting around to visit their patients. About 20 veterinary colleges were in existence, of which all but five have closed their doors. These institutions graduated 213 veterinarians in their 1898 classes. The outlook that year was anything but bright, as horses were selling for extremely low prices. The market prices for other classes of live stock did not enter into the picture. Poultry practice had not even been dreamed of and, with few exceptions, canine practice was limited to the spaying of bitches.

We will rest content with having made very brief references to a few of the changes that have taken place during the past 39 years, and with having mentioned some of the advantages enjoyed by the veterinarian of 1937, which were not available to his colleagues of 1898. It occurs to us in passing that the latter were more fortunate, in some respects, at least. They did not have to contend with many of the aggravations of more recent times. However, few of us are privileged to enjoy the pure gold without the dross. On to Omaha. The program for the meeting is outlined on the following pages. It reflects, in large measure, the advances made since we met in Omaha in 1898.

HOTEL FONTENELLE, OMAHA, HEADQUARTERS OF THE 1937 CONVENTION.

WHEN TO MEET

A short time ago, we made some comments on the possible desirability of selecting the cities for our annual meetings two years in advance, rather than one year ahead, as has been customary heretofore. The reactions to this suggestion have been favorable in the majority of cases. It can be said that no serious objections to the suggested change have been registered. However, one thought usually brings on another, and such has been the case in the present instance. We can get at the next thought by repeating a question that has been asked by at least a dozen members who have taken the time to express themselves. Here it is: Why are A. V. M. A. meetings usually held in August?

Most of us are creatures of habit and perhaps this will explain, in part at least, why we usually hold our convention in August. If there should be any doubt as to the truthfulness of the statement that we are in the habit of convening in August, just let us look at the record. Starting with the meeting held in Oakland, California, in 1915, we have met 18 times during August, once in July (Portland, Oregon, 1925), twice in September (Denver, 1921, and Phladelphia, 1927), and once in November (New Orleans 1919). Section 1 of Article IV of the Constitution provides that the regular annual meeting shall be convened not earlier than July 15, nor later than December 31. So much for the facts in the case.

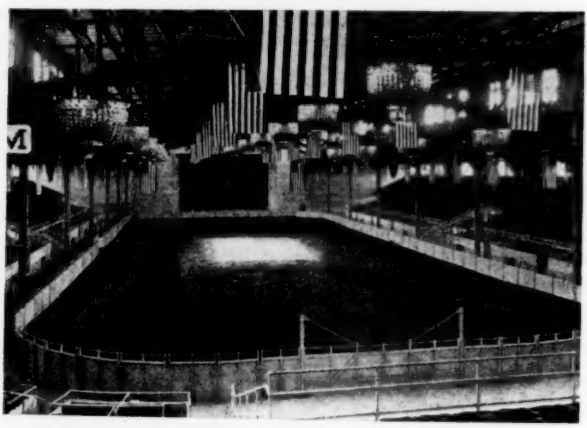
There probably are several reasons that have operated in the selection of meeting dates. For the past 15 years, at least, the Executive Board has been guided very largely by the recommendations of each succeeding committees on local arrangements. Quite often it has been necessary to steer clear of some other large meeting in the city where we were to meet, but usually this has meant shifting the dates only a week in one direction or another. The colleges undoubtedly have been an important factor in the consideration of meeting dates. Members of college faculties find it difficult to get away after classes are back in the fall. Then, members having children of school age find it inconvenient to leave home for a week after schools have opened in September, especially those members who are in the habit of taking their wives with them.

It is a foregone conclusion that it will be impossible to select a time that will suit everybody. Therefore, it becomes a matter of pleasing the greatest number possible. The problem is not an easy one to solve, as there are a number of factors which merit serious consideration. If there is to be any change, we do not want to jump out of the frying-pan into the fire. August

conventions, with all of the disadvantages charged against them (hot weather, busy times for practitioners, conflicts with state fairs, and several others), have been drawing crowds, and Omaha promises to be no exception.

In some of the informal gatherings at which this question has been discussed, the point has been raised that the meetings which are held in Chicago around the first of December each year, at the time of the International Live Stock Exposition, always attract several hundred veterinarians from all parts of the country. The point is taken, not necessarily to advance the interests of Chicago as a meeting place, but to show that many veterinarians will travel a considerable distance to attend a meeting in December.

There is one disadvantage of the August convention that has not received much consideration, probably because it is of comparatively recent origin. It relates to the A. V. M. A. House of Representatives. Most state associations have winter meetings, but quite a few do not have summer meetings (Illinois, Indiana, Iowa, Kansas, Nebraska, Ohio, just to mention a few). One of the objects of the formation of the House of Representatives was to bring about a closer tie-up of the state associations with the parent body. With the great majority of the state associations meeting during the months of January and February, would there not be considerable advantage in having the A. V. M. A. convention held later in the year than at present, say in November or December, just before the state associations meet? Or would it be better to have the A. V. M. A. meet soon after the state associations have met? Think it over.



INTERIOR AK-SAR-BEN COLISEUM, WHERE THE CLINIC WILL BE HELD.

TO BAN ALL DOGS

Somebody is always trying to take the joy out of life. This time it started in New York City, where, according to reports, a movement has been started to ban all dogs from the city. We hope that the report is groundless, but it is stated that a society has been formed in Manhattan for the expressed purpose of exiling all canines from the metropolis. Well, some big ideas have been born in New York, but this one gets the leather medal. To add insult to injury, "dog-lovers" are being given the "credit" for having conceived this wonderful idea, ostensibly in the best interests of our canine friends, because they "would be better off in the country." Reports available do not indicate whether it is a part of the plan to banish the owners along with their dogs.

Not being satisfied with having "started something" in the interests of man's best friend, the sponsors of this latest of inane ideas evidently felt the need for something to be used as a reason for their appeal, so they have accused the dog of being a spreader of disease. Well, it is not the first time that somebody has tried to hang this undeserved accusation on the dog. Outside of rabies, there is not a single disease of any importance that is spread by dogs. In passing, we might say that whenever the authorities decide to unscramble the now divided responsibility of controlling rabies and turn this animal-disease problem over to the veterinary profession, where it belongs, an outbreak of rabies will rate a headline in our newspapers for one reason only—its extreme rarity.

It is to be hoped that the idea of the dog ban will die aborning. There are about one hundred persons killed every day in the year by automobiles throughout the United States. Why not pass a law making it illegal for anybody to drive an automobile on a public highway? Such a law would save more lives than banishing all dogs from *all* of our large cities.

THE RABIES MENACE

"The Rabies Menace" has become a common headline in medical, veterinary and secular literature. For example, the *Journal* of the American Medical Association (May 22, 1937) makes use of that headline for its leading editorial. Assuredly, a medical problem that "makes" that lieu is a medical problem indeed. From the thoughtful veterinarian's point of view, the disconcerting thing about these articles is that the veterinary service

is never mentioned among the forces invoked to step in and curb the increasing incidence of the disease.

The editorial (*ad hoc*), after citing the high mortality from rabies in this country as compared with that of European countries, cries out for help from what is called the "machinery of social organization," comprising the press, the public health officials, the physicians and the animal humane societies. There is neither mention nor hint that the problem is first and last a problem of the veterinary service—the service presumably charged with the handling of animal diseases. The significance of the omission is that the veterinary profession has not yet been accorded a place in the "machinery of social organization" required to check the spreading of rabies.

True, we have a federal bureau, state services, and an army of officially accredited clinicians charged with the suppression of contagious diseases of animals, but, obviously, no one, even in the upper cadre of epidemiologists, has yet been impressed with the fact, or with the idea of developing and recognizing these forces and using them for the benefit of mankind when critical situations arise.

It would seem sarcastic to say that someone should tell folks about the veterinary profession were it not for the fact that the veterinary service is securely hidden away under such ill-defined names as bureaus of animal industry, state regulatory services, live stock sanitary boards, meat and milk inspection divisions, animal pathology departments, and the like, with the word "veterinarian" so beautifully deleted that even the best informed epidemiologists know not where to look for a policeman to help out when animal diseases show an ugly front. So, it is not surprising that the aid of the press, the physicians and the humane societies is invoked to control rabies. The veterinary service, laboring under its multiplicity of confusing names, is not yet regarded as a unit of the "machinery of social organization." The fault is our own. The word "veterinary" has been so generally suppressed in this country since the turn of the century that it has lost connection with problems of national importance.

L. A. M.

WERE ANY OF THEM VETERINARIANS?

In the foregoing editorial, L. A. M. directs attention to a well-defined trend that has not been fully appreciated by most members of our profession. Regardless of what may have been the purpose—probably not the same in all cases—there has been

a very noticeable increase in the use of names and titles that have displaced the words "veterinary" and "veterinarian." L. A. M. did not mention all of them. Right here in Illinois, for example, veterinarians in the employ of the State Department of Agriculture are commissioned as "T. B. Inspectors." What of the scores of institutions throughout the country that are conspicuously advertised to the public as "animal hospitals"? In this connection the following incident comes to mind:

Several years ago, a feature writer called to get some information on the veterinary profession, to be used in an article that was to be given rather wide distribution. We supplied a lot of facts and figures and then suggested that several veterinary hospitals be visited for the purpose of getting some first-hand information on this important phase of modern veterinary work. We were promised that this would be done. About a week later, the feature writer returned for some more data. We asked him whether he had visited any of the hospitals suggested. He said that he had and then proceeded to relate some of his impressions, with particular reference to one of the hospitals. It was so well conducted, so well appointed, so clean, so quiet, and a few other adjectives. But imagine our discomfiture when he asked the question: "Were any of those good-looking young doctors in white suits veterinarians?"

EXECUTIVE BOARD ELECTIONS

There will be no change in the personnel of the Executive Board of the A. V. M. A. as a result of the regular elections held in two districts this year. These elections came to a close during the past month after sixty days of unusually heavy balloting. In District 1 (Canada), Dr. A. E. Cameron, the present incumbent, was re-elected for another five-year term, after a close race. Seventy-five per cent of the Canadian members who were eligible to vote cast their ballots.

In District 9 (New York and the New England States), where almost 500 members are located, unusual interest was taken in the election. About 63 per cent of the members who were entitled to vote in this district exercised their privilege, with the result that Dr. Harry W. Jakeman, of Boston, received the highest number of votes and will succeed himself for the term 1937-1942.

In the special election held in District 10 (Michigan and Ohio), to select a successor to Dr. Oscar V. Brumley, who resigned as a district member of the Executive Board following his election

as president-elect of the A. V. M. A. at Columbus, Dr. F. A. Zimmer, State Veterinarian of Ohio, has been elected to fill out the unexpired term. President Foster made no temporary appointment in District 10, as he considered the district well represented by Dr. Brumley, who serves on the Board, both this year and next, as a member ex-officio. Dr. Zimmer will sit on the Board at the annual meeting in Omaha next month.

THE CONVENTION PROGRAM

Following the custom of recent years, we are presenting in this issue of the JOURNAL a tentative list of the addresses and papers which will constitute the literary program for the Omaha convention. The officers of some of the sections got an unusually early start this year and as a result were able to complete the arrangement of the programs for their sections almost two months in advance of the opening date of the convention.

A slight change in the arrangement of the program, as compared with recent years, has been decided upon for the Omaha meeting. All of the sectional meetings will be held on Wednesday and Thursday, using both mornings and afternoons for these meetings. The only general session of the convention, in addition to the opening session, will be held Tuesday afternoon. At this session will be five outstanding addresses followed by a short business session for the nomination of officers. On another page in this issue will be found a skeleton outline of the program for the week. The list of addresses and papers (tentative) follows:

General Session

Food Inspection for the Civilian Conservation Corps—Hon. Robert Fechner, Director, Emergency Conservation Work, Washington, D. C.

The Public Health Program Under Title VI of the Social Security Act—Dr. C. E. Waller, Assistant Surgeon General, United States Public Health Service, Washington, D. C.

What Can Be Expected from a General Campaign Against Tuberculosis—Dr. John F. Allen, Associate Professor of Medicine, University of Nebraska College of Medicine, Lincoln, Nebr.

Greater Achievement Through Closer Coöperation—Dr. John R. Mohler, Chief, U. S. Bureau of Animal Industry, Washington, D. C.

Organizing a Bureau of Animal Welfare for Kwangsi Province, Southwest China—Dr. E. A. Rodier, Pullman, Wash.

Section on General Practice

Chairman: Dr. S. E. Hershey, Charleston, W. Va.
Secretary: Dr. C. C. Hastings, Williamsville, Ill.

"Dietary Deficiencies and Related Symptomatology in Domestic Animals,"—Dr. George H. Hart, University of California, Davis, Calif.

"Endocrines and Their Rôle in Reproduction,"—Dr. W. G. Venzke, University of Wisconsin, Madison, Wis.

"Artificial Insemination,"—Dr. H. E. Kingman, Wyoming Hereford Ranch, Cheyenne, Wyo.

"Some Hernias in the Equine and Bovine,"—Dr. E. R. Frank, Kansas State College, Manhattan, Kan.

"Diseases of Beef and Feeder Cattle,"—Dr. I. J. Kleaveland, Sioux Rapids, Iowa.

"Early Research and Present-Day Problems in Hog Cholera Immunization,"—Dr. Henry Hell, Wilton Junction, Iowa.

"Urinary Calculi with Special Reference to Cattle and Sheep,"—Dr. I. E. Newsom, Colorado State College, Fort Collins, Colo.

"The Diagnosis and Treatment of Equine Arthritis,"—Dr. Cassius Way, New York, N. Y.

"Bang's Disease from a Practitioner's Viewpoint,"—Dr. S. L. Stewart, Olathe, Kan.

"Intravenous Use of Sodium Iodide in Actinomycosis,"—Dr. James Farquharson, Colorado State College, Fort Collins, Colo.

Section on Sanitary Science and Food Hygiene

Chairman: Dr. C. C. Hisel, Oklahoma City, Okla.
Secretary: Dr. I. A. Merchant, Ames, Iowa.

Milk Inspection—Dr. W. N. Martin, El Reno, Okla.

Brucellosis in the Middle West—Dr. Carl F. Jordan, Director of Bureau of Communicable Disease, Iowa State Department of Health, Des Moines, Iowa.

Meat Inspection—Dr. W. H. Lawrence, Oklahoma City, Okla.

Inspection and Grading of Animal Products Under the Bureau of Agricultural Economics—Dr. R. B. Mericle, Omaha, Neb.

The Supervision of the Milk Supply for an Iowa Institution (illustrated with motion pictures)—Dr. W. H. Riser, Glenwood, Iowa.

How the Veterinarian Can Coöperate in Preventing Losses in the Shipment of Live Stock—Mr. Earle G. Reed, Chairman, National Live Stock Loss Prevention Board, Omaha, Neb.

Section on Research

Chairman: Dr. B. A. Beach, Madison, Wis.

Secretary: Dr. H. C. H. Kernkamp, Saint Paul, Minn.

Further Observation on Oat Hay Poisoning—Dr. Frank Thorp, Colorado State College, Fort Collins, Colo.

Hemorrhagic Septicemia Vaccination Related to the Bang's Agglutination Reaction—Drs. C. H. Kitselman and H. F. Lienhardt, Kansas State Agricultural College, Manhattan, Kans.

Some Effects on Cattle with Vaccination with *Brucella Abortus* with United States Bureau of Animal Industry Culture No. 19—Dr. C. M. Haring, University of California, Berkeley, Calif.

The Study of the Vaginal Contents of Pregnant, Bang-Infected Cows for the Presence of *Brucella Abortus*—Dr. C. P. Fitch, Dr. W. L. Boyd and Lucille M. Bishop, University of Minnesota, Saint Paul, Minn.

Some Nutritional Studies of Swine—Dr. H. E. Biester, Department of Veterinary Investigation, Iowa State College, Ames, Iowa.

An Investigation of Severe Losses from Abortion in Ewes—Drs. A. M. Lee and L. H. Scrivner, University of Wyoming, Laramie, Wyo.

Studies on *Erysipelothrix Rhusiopathiae* with Special Reference to Smooth and Rough Type Cultures—Drs. H. W. Schoening, W. S. Gochenour and C. G. Grey, United States Bureau of Animal Industry, Washington, D. C.

Moldy Corn Poisoning in Horses—Dr. L. H. Schwarte, Veterinary Research Institute, Iowa State College, Ames, Iowa.

Section on Small Animals

Chairman: Dr. John F. Planz, Akron, Ohio.

Secretary: Dr. Helen Richt Irwin, Tulsa, Okla.

Nervous Diseases of Dogs—Dr. C. F. Schlotthauer, Mayo Foundation, Rochester, Minn.

Non-parasitic Skin Diseases of the Dog—Dr. S. W. Haigler, Saint Louis, Mo.

Blood Transfusions in Dogs—Dr. Chas. Bower, Topeka, Kan.

General Anesthesia for Small Animals—Dr. E. E. Sweebe, North Chicago, Ill.

Cat Diseases Found in Practice—Dr. F. F. Parker, Des Moines, Iowa.

Uterine Inertia—Dr. F. Perrin, Lincoln, Neb.

The Place of the X-Ray in Small-Animal Practice—Dr. G. S. Elwood, Chicago Ill.

Section on Poultry

Chairman: Dr. P. V. Neuzil, Blairstown, Iowa.

Secretary: Dr. F. D. Patterson, Jr., Auburn, Ala.

Diagnosis of Poultry Diseases—Dr. S. H. McNutt, Iowa State College, Ames, Iowa.

Avian Tuberculosis—Dr. A. J. Durant, University of Missouri, Columbia, Mo.

Poultry Mortality of Non Infectious Origin—Dr. R. E. Lubbehusen, University of California, Berkeley, Calif.

Controlling Poultry Diseases—Dr. F. C. Tucker, Claypool, Ind.

Transmission of Fowl Leukosis, Using Filtrates at Various pH Levels—Dr. C. D. Lee, Iowa State College, Ames, Iowa.

Coli-Bacillosis in Young Chicks—Dr. C. R. Davis, Maryland State Board of Agriculture, Live Stock Sanitary Service Laboratory, College Park, Md.

The Viability and Immunizing Value of Egg-Propagated Laryngotracheitis Virus—Dr. F. R. Beaudette and Dr. C. B. Hudson, New Jersey Agricultural Experiment Station, New Brunswick, N. J.

Fowl Leukosis—Dr. E. L. Stubbs, University of Pennsylvania, Philadelphia, Pa.

Pullorum Disease in Turkeys—Studies on Incidence and Carriers—Dr. W. R. Hinshaw, University of California, Davis, Calif.

The Clinic

Dr. J. E. Weinman, of Lincoln, Neb., is chairman of the committee in charge of the Clinic which will be held at the Ak-Sar-Ben Coliseum, on Friday, August 20. The program for the Clinic had not been completed at the time of going to press, but Dr. Weinman advises that the Clinic will be divided into a number of sections, as has been the general plan of recent A. V. M. A. clinics.

In the Bovine Section, mastitis will be given a prominent place and demonstrations of cyanide poisoning are being arranged. The tentative program for the Equine Section includes

(Continued on page 121)

CONDENSED PROGRAM

Monday, August 16

MORNING	Committee meetings.
AFTERNOON	Executive Board meeting.
EVENING	First session, House of Representatives.

Tuesday, August 17

MORNING	Opening session, Ball Room. Ladies attend.
AFTERNOON	General session, Ball Room. Addresses. Business session for nomination of officers. Reception and tea for ladies, Aquila Court.
EVENING	Polo game and den show, Ak-Sar-Ben Coliseum.

Wednesday, August 18

MORNING	Sectional meetings: General Practice. Research. Sanitary Science and Food Hygiene. Complimentary breakfast and meeting of Ladies' Auxiliary, Omaha Atheltic Club.
AFTERNOON	Sectional meetings: Small Animals. Poultry. Conference of Extension Veterinarians.
EVENING	President's reception, general banquet and dance.

Thursday, August 19

MORNING	Sectional meetings: General Practice. Sanitary Science and Food Hygiene. Semi-annual meeting (business session), American Animal Hospital Association.
NOON	Luncheon and style show for ladies, Paxton Hotel. Luncheon-meeting of National Association of Chief Live Stock Officials.
AFTERNOON	Sectional meetings: Small Animals. Research-Poultry.
EVENING	Alumni dinner-meetings. House of Representatives. Closing session, Ball Room.

Friday, August 20

MORNING	Clinic, Ak-Sar-Ben Coliseum.
NOON	Luncheon for ladies, Fontenelle Hotel, and trip to the Joslyn Memorial Art Museum. Program of classical music.
AFTERNOON	Clinic continued.

Omaha Hotels

Hotel	Address	Distance from Headquarters (Blocks)	Rooms	Rates			
				Without Bath		With Bath	
				Single	Double	Single	Double
Fontenelle (Official headquarters)	18th and Douglas		400	...	...	$2.50–$5.00	$4.00–$7.50
Paxton	14th and Farnam	6	320	...	...	$2.50 up	$4.00 up
Conant	16th and Harney	4	250	...	...	$2.00–$3.00	$3.00–$4.00
Flatiron	17th and Saint Mary Ave.	4	100	$1.00–$1.50	$1.50–$2.50	$1.75–$2.50	$2.50–$3.50
Castle	16th and Jones	7	275	$1.00	$1.50–$2.00	$1.50–$2.50	$2.50–$3.50
Keen	18th and Harney	2	100	$1.25–$1.50	$2.00–$2.50	$1.50–$2.50	$2.50–$3.50
Loyal	16th and Capital	4	150	$1.25–$1.50	$1.50–$2.50	$2.00–$3.00	$2.50–$5.00
Sanford	19th and Farnam	2	200	$1.50–$1.75	$2.00–$2.50	$2.00 up	$3.00 up
Wellington	18th and Farnam	1	150	$1.25–$1.50	$2.00–$2.50	$2.00–$2.50	$3.00–$3.50
Carlton	15th and Howard	6	125	$1.00	$1.50	$1.50	$2.00
Hamilton	24th and Farnam	7	125	...	...	$1.50–$2.00	$2.00–$3.00
Rome	16th and Jackson	6	250	$1.50	$2.50	$2.00–$3.50	$3.50–$5.50
Logan	18th and Dodge	1		...	...	$2.50–$6.00	$3.50–$7.50

APPLICATIONS FOR MEMBERSHIP

This month we are giving first listing to 87 applications for membership, the largest number to be filed during any one month since August, 1929, when 97 applications were received. The total number of applications filed during the ten months ended June 30 reach 384. Of this number, 45 are credited to Iowa and 31 to Nebraska, the two states that are most vitally interested in the 1937 convention. A considerable part of the attendance at Omaha will be drawn from these two states, so it is not hard to find a reason for the large number of applications being filed by veterinarians throughout Nebraska and Iowa.

Several states far removed from the convention city have done conspicuously well in contributing to the 384 total. California, always a contender for high honors in membership campaigns, has 26 to her credit. Then comes Pennsylvania with 21. New York and Texas are tied with 20 each. Florida has contributed 19, and Minnesota and Ohio show with 18 for each state. Illinois follows with 14, Michigan with 13, and Alabama with 11.

As has been the custom every six months, we quote Section 1 of Article 2 of the By-laws, covering the manner of filing an application for membership:

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

AHLERS, FRED R. — La Motte, Iowa
D. V. M., Iowa State College, 1902
Vouchers: P. V. Neuzil and N. L. Nelson.

ANDERSON, ALFRED A. — 518 W. 3rd St., Grand Island, Neb.
D. V. S., Kansas City Veterinary College, 1909
Vouchers: Paul L. Matthews and E. C. Jones.

ANDERSON, THOMAS E. Bedford, Iowa
M. D. C., Chicago Veterinary College, 1907
Vouchers: A. C. Drach and R. L. South.

ANDREWS, WILLIAM L. Milton, Iowa
D. V. M., Saint Joseph Veterinary College, 1921
Vouchers: H. T. Corbet and P. V. Neuzil.

ARMSTRONG, JAMES W. 95 Eldridge St., Cranston, R. I.
D. V. M., Ohio State University, 1933
Vouchers: W. F. Guard and Walter R. Hobbs.

ARMSTRONG, WILLIAM H. 601 S. Elena Ave., Redondo Beach, Calif.
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and Walter R. Krill.

BAER, J. W. Malvern, Iowa
D. V. M., Kansas City Veterinary College, 1912
Vouchers: George Wessels and A. C. Drach.

BAIRD, NORMAN G. Sanford Veterinary Hospital, Sanford, N. C.
D. V. M., Alabama Polytechnic Institute, 1937
Vouchers: I. S. McAdory and F. D. Patterson.

BARTENSLAGER, ARTHUR V. Stewartstown, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: R. S. Amadon and A. Henry Craige, Jr.

BEARD, R. V. 2535 Mount Pleasant Rd., Burlington, Iowa
D. V. M., Kansas City Veterinary College, 1918
Vouchers: P. V. Neuzil and Grant B. Munger.

BLANK, LLOYD C. Emerson, Iowa
D. V. M., Iowa State College, 1931
Vouchers: Paul L. Matthews and J. A. Lueth.

BOWSTEAD, LEONARD A. Dewitt, Iowa
B. Sc., Iowa State College, 1933
D. V. M., Iowa State College, 1937
Vouchers: T. S. Leith and Chas. Murray.

BOYLE, WILLIAM H. 932 Cochran Ave., Schuyler, Neb.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: S. W. Phillips and Jno. D. Sprague.

BRADY, EZRA M. 708 E. 9th St., Atlantic, Iowa
D. V. M., Iowa State College, 1912
Vouchers: Paul L. Matthews and J. A. Lueth.

BROMWELL, V. G. Center Point, Iowa
D. V. M., Iowa State College, 1918
Vouchers: W. C. Vollstedt and T. W. Munce.

BUCHLEITER, WALTER H. Coin, Iowa
D. V. M., Kansas City Veterinary College, 1916
Vouchers: George Wessels and A. C. Drach.

BUCKLEY, HOWARD P. c/o Dr. G. S. McKee, Beaver Falls, Eastvale, Pa.
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and Walter R. Krill.

CAIRNS, GEORGE 464 Bathurst St., Toronto, Ont.
B. V. Sc., Ontario Veterinary College, 1925
M. R. C. V. S., Royal College of Veterinary Surgeons (Edinburgh), 1926
Vouchers: J. A. Campbell and C. D. McGilvray.

CHAMBERS, CHARLES H. 304 N. Platte Ave., Fremont, Neb.
D. V. M., Chicago Veterinary College, 1920
Vouchers: Paul L. Matthews and W. T. Spencer.

COLLINS, C. M. Route 3, Ottumwa, Iowa
D. V. M., Iowa State College, 1915
Vouchers: P. V. Neuzil and N. L. Nelson.

COX, FORREST O. Hillsville, Va.
D. V. M., Kansas State College, 1934
Vouchers: H. L. Lyon and H. S. Miller.

CRAIGE, JOHN E. 1317 S. 57th St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: R. S. Amadon and E. T. Booth.

CRIDER, CLAYTON L. Elkader, Iowa
D. V. M., Iowa State College, 1914
Vouchers: P. V. Neuzil and John B. Bryant.

CRISMAN, DAVID W. 236 Manoa Rd., Brookline, Upper Darby, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: R. S. Amadon and A. Henry Craige, Jr.

DAPPEN, ROY R. Brooklyn, Iowa
D. V. M., Kansas City Veterinary College, 1917
Vouchers: W. C. Verploeg and H. T. Corbet.

ELLIOT, THOMAS A. State House, Boise, Idaho
D. V. M., State College of Washington, 1914
Vouchers: George F. Motteler and E. M. Gildow.

FARR, ERNEST Montezuma, Iowa
D. V. S., Kansas City Veterinary College, 1910
Vouchers: H. T. Corbet and T. W. Munce.

FITCH, W. H. Walcott, Iowa
B. V. Sc., Ontario Veterinary College, 1917
Vouchers: P. V. Neuzil and W. C. Voolstedt.

FULLER, JENNINGS B. Huntley, Wyo.
D. V. M., Colorado State College, 1924
Vouchers: G. D. Anderson and H. D. Port.

GERLACH, PHILIP H. 25—8th Ave., Laurel Beach, Milford, Conn.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and R. S. Amadon.

GUBSER, K. H. Adel, Iowa
D. V. M., Iowa State College, 1920
Vouchers: N. L. Nelson and H. T. Corbet.

HELWIG, JOHN H. Box 233, Reynoldsburg, Ohio
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and R. E. Nichols.

HESSE, CHARLES P. 45 W. 9th Ave., Columbus, Ohio
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Walter R. Hobbs.

HOWE, NELSON S., JR. 15 S. 3rd St., Niles, Mich.
D. V. M., Michigan State College, 1935
Vouchers: M. J. Court and Edw. K. Sales.

INGMAND, JOSEPH E. Red Oak, Iowa
M. D. C., Chicago Veterinary College, 1906
Vouchers: Paul L. Matthews and J. A. Lueth.

JACKSON, W. CLINTON 231 N. Sandusky St., Bucyrus, Ohio
D. V. M., Ohio State University, 1937
Vouchers: F. B. Jackson and W. F. Guard.

KEEFER, WESLEY O. 50 E. Buchtel Ave., Akron, Ohio
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and C. R. Donham.

KEELER, HUGH H. Route 1, Milan, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and C. J. Marshall.

KELLY, ERNESTINE G. 400 Winston Ave., Govans, Baltimore, Md.
D. V. M., Ohio State University, 1937
Vouchers: Jas. D. Grossman and W. F. Guard.

KERN, ELLIS W. 534 W. Elsmere Place, San Antonio, Texas
D. V. M., Kansas State College, 1914
Vouchers: Matthew E. Gleason and Chas. W. Neal.

KINTON, SPERRY C. Warren, Pa.
V. M. D., University of Pennsylvania, 1936
Vouchers: A. Henry Craige, Jr., and G. A. Dick.

LACROIX, LEON J. Box 445, Evanston, Ill.
Ph. C., University of Southern California, 1929
D. V. M., Cornell University, 1937
Vouchers: E. C. Khuen and H. Preston Hoskins.

LANPHERE, LYSLE J. Kimball, Neb.
D. V. M., Colorado State College, 1927
Vouchers: E. C. Jones and Carl J. Norden.

LEACH, LT. BENJAMIN F. Camp McCoy, Sparta, Wis.
B. V. Sc., V. S., Ontario Veterinary College, 1935
Vouchers: Maj. S. A. Grover and Maj. Will C. Griffin.

LOILER, ROY A. Sidney, Iowa
D. V. M., Chicago Veterinary College, 1912
Vouchers: Paul L. Matthews and J. A. Lueth.

MAHER, LOUIS J. 106 N. 5th Ave., Highland Park, N. J.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and G. A. Dick.

MARSH, EDWARD T. 521 S. Madison St., Waupun, Wis.
B. A., University of Wisconsin, 1932
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and C. R. Donham.

MAXFIELD, FRED M. Gilman, Iowa
D. V. M., Kansas City Veterinary College, 1914
Vouchers: W. C. Verploeg and H. T. Corbet.

MCCREARY, ANDREW J. Escambia County Health Dept., Pensacola, Fla.
D. V. M., Alabama Polytechnic Institute, 1935
Vouchers: Robert M. Parker and E. L. Reed.

MCELYEA, LEW W. Ames, Iowa
D. V. M., Iowa State College, 1911
Vouchers: N. L. Nelson and T. W. Munce.

MILLARD, PHILIP S. 3625 Rucker Ave., Everett, Wash.
D. V. M., Colorado State College, 1933
Vouchers: V. C. Pauhlman and S. S. Worley.

MOSLANDER, WILLIAM J. 518 W. 3rd St., Grand Island, Neb.
D. V. M., McKillip Veterinary College, 1919
Vouchers: Paul L. Matthews and E. C. Jones.

MOYER, JESSE B. Orwigsburg, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: R. S. Amadon and A. Henry Craige, Jr.

MUENCH, WALTER P. Mapleton, Minn.
D. V. M., Iowa State College, 1934
Vouchers: M. W. Ray and E. H. Enge.

MYERS, THEODORE R. Wilbur, Wash.
D. V. M., Colorado State College, 1931
Vouchers: W. P. Guard and I. E. Newsom.

OLDHAM, CARL M. Charlottesville, Ind.
D. V. M., Ohio State University, 1937
Vouchers: W. F. Guard and Walter R. Krill.

OSBURN, DONALD C. Auburn, Iowa
D. V. M., Iowa State College, 1934
Vouchers: George Wessels and A. C. Drach.

OVERBAUGH, A. N. Hull, Iowa
D. V. M., Saint Joseph Veterinary College, 1923
Vouchers: J. E. Weinman and T. W. Munce.

PAYEN, LOUIS J. Truckee, Calif.
B. S., University of California, 1934
D. V. M., Ohio State University, 1937
Vouchers: Jas. D. Grossman and W. F. Guard.

PECK, EUGENE, W. Auburn, Neb.
D. V. M., Kansas State College, 1933
Vouchers: Paul L. Matthews and W. T. Spencer.

POWELL, GLENN J. 9 Mill St., Nunda, N. Y.
D. V. M., Cornell University, 1919
Vouchers: W. A. Hagan and J. G. Wills.

PRESTON, EDWARD C. Deal, N. J.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr., and R. S. Amadon.

PURSE, WILLIAM J. 8043 Wornall Rd., Kansas City, Mo.
D. V. M., Colorado State College, 1932
Vouchers: Carl H. Schulz and O. F. Reihart.

RABE, OTTO A. Winslow, Ill.
D. V. M., McKillip Veterinary College, 1915
Vouchers: C. W. Swingley and Chas. H. Rosenstiel.

ROLSTON, E. A. 2103 Harrison Dr., Clinton, Iowa
D. V. M., Iowa State College, 1921
Vouchers: P. V. Neuzil and J. J. Strandberg.

RYDELL, ROBERT O. Wheaton, Minn.
D. V. M., Iowa State College, 1934
Vouchers: Robert A. Merrill and Charles E. Cotton.

SCHAEFER, GEORGE L. Tekamah, Neb.
D. V. M., Kansas City Veterinary College, 1913
D. V. M., Cornell University, 1916
Vouchers: Paul L. Matthews and W. T. Spencer.

SCHOFIELD, WILLIAM C. Louisiana State University, Baton Rouge, La.
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and Walter R. Krill.

SCHUCK, WILLIAM J., JR. 2441 N. 8th St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: R. S. Amadon and A. Henry Craige, Jr.

SCOTT, DAVID C. Tekamah, Neb.
D. V. S., Kansas City Veterinary College, 1905
Vouchers: Paul L. Matthews and W. T. Spencer.

SEARS, KIRTLEY 508 N. Buchanan St., Maryville, Mo.
D. V. M., Saint Joseph Veterinary College, 1923
Vouchers: A. C. Drach, R. L. South and A. T. Kinsley.

SENIOR, GEORGE B. Creston, Iowa
B. V. Sc., Ontario Veterinary College, 1921
Vouchers: N. L. Nelson and P. V. Neuzil.

SOLLOMI, PHILLIP A. 12522 Saint Clair Ave., Cleveland, Ohio
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Walter R. Hobbs.

STADLER, ROBERT J. 143 Kelsey St., New Britain, Conn.
V. M. D., University of Pennsylvania, 1937
Vouchers: A. Henry Craige, Jr. and C. J. Marshall.

STEWART, HUGH H. Kanawha, Iowa
D. V. M., Kansas City Veterinary College, 1913
Vouchers: J. E. Weinman and T. W. Munce.

STONE, WINFIELD S. New York State Veterinary College, Ithaca, N. Y.
D. V. M., Cornell University, 1935
M. S., Cornell University, 1936
Vouchers: Herbert L. Gilman and R. R. Birch.

SYPIEN, ADOLF J. 8241 S. Justine St., Chicago, Ill.
D. V. M., Michigan State College, 1936
Vouchers: E. C. W. Schubel and Ward Giltner.

TICE, ALBERT K. 7 Wheeler Ave., Cortland, N. Y.
D. V. M., Cornell University, 1935
Vouchers: John L. McAuliffe and E. V. Moore.

TROMPETER, LEE ROY 701 West St., Rapid City, S. Dak.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: Carl J. Norden and C. H. Hays.

TYNER, H. E. New London, Iowa
M. D. C., Chicago Veterinary College, 1911
Vouchers: W. C. Vollstedt and P. V. Neuzil.

VON KAENEL, FRED 117 W. Southern Ave., Bucyrus, Ohio
D. V. M., Ohio State University, 1924
Vouchers: C. H. York and W. F. Guard.

WALLACE, MYRON F. 210 Central Ave., Kansas City, Kan.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: H. B. Hickman and O. F. Reiha[illegible].

WESSON, HARRISON R. Jefferson, Iowa
D. V. M., Chicago Veterinary College, 1913
Vouchers: R. L. South and A. C. Drach.

WILEY, CHARLES W. Farson, Iowa
D. V. S., Kansas City Veterinary College, 1910
Vouchers: C. R. Fry and F. F. Meads.

WOOD, STANLEY N. 609—7th St., Ames, Iowa
D. V. M., Iowa State College, 1936
Vouchers: T. S. Leith and Chas. Murray.

WOODS, LAIRD Malcom, Iowa
D. V. M., Kansas City Veterinary College, 1912
Vouchers: A. H. Quin, Jr. and T. W. Munce.

ZIMMERMAN, IRVING 138 W. 83rd St., New York, N. Y.
D. V. M., Cornell University, 1935
Vouchers: George Watson Little and Frank Bloom.

Applications Pending

SECOND LISTING

(See June, 1937, JOURNAL)

Akin, Joe E., 703 Nebraska St., Sioux City, Iowa.
Anderson, Rolland, Box 175, Worland, Wyo.
Anstey, John A., Massena, Iowa.
Bond, Thomas L., Cumberland, Iowa.
Boone, Charles W., R. 2 Roanoke, Va.
Bunker, John W., Winterset, Iowa.
Campbell, Harry C., 2300 Locust St., Philadelphia, Pa.
Crall, William W., Pioneer, Ohio.
Denham, William B., Cumberland, Iowa.
Ditman, Noran L., Winside, Neb.
Garrett, Harley U., Saint Charles, Iowa.
Gehrman, Frederick S., 5324 Lyndale Ave. S., Minneapolis, Minn.

Hage, Dennie M., Ida, Mich.
Halver, Dale L., Shakopee, Minn.
Hansen, Frederic W., Pelican Rapids, Minn.
Hodder, Charles P., 19426 Troy Place, Detroit, Mich.
Hunsberger, Lt. Ray S. Philippine Dept., U. S. Army, Manila, P. I.
Jeffers, Boyd, 355 Walnut St., Lexington, Ky.
Laughlin, Newton C., 1520 Parker Drive, Mayfield Heights, South Euclid, Ohio.
McCormick, Hugh C., 821 Wealthy St. S. E., Grand Rapids, Mich.
McQuistion, Ray B., Trout Creek, Mich.
Myers, Philip, 809 Flores Ave., Laredo, Texas.
Navarro, Manuel, 527 University Ave., Saint Paul, Minn.
Nelms, Emery E., Smithfield, N. C.
Roche, Johnson R., Gowrie, Iowa.
Smith, William R., Box 1365, Las Vegas, Nev.
Sprague, Arthur W., 519 Live Stock Exchange Bldg., Omaha, Neb.

The amount which should accompany an application filed this month is $7.50, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

New York State Veterinary Medical Association. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. July 7-9, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Northwestern Indiana Veterinary Medical Association. Dr. G. W. Andree's office, Rensselaer, Ind. July 8, 1937. Dr. G. W. Andree, Secretary, Rensselaer, Ind.

Georgia State Veterinary Medical Association. Columbus, Ga. July 8-9, 1937. Dr. J. E. Severin, Secretary, 357 Edgewood Ave. S. E., Atlanta, Ga.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. July 12, 1937. Dr. W. H. Riser, Secretary, Glenwood, Iowa.

Willamette Valley Veterinary Medical Association. (Joint meeting with Northwest Veterinary Medical Association.) Multnomah Hotel, Portland, Ore. July 12-14, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical associations.) Multnomah Hotel, Portland, Ore. July 12-14, 1937. Dr. B. T. Sims, Secretary, Oregon State College, Corvallis, Ore.

San Diego County Veterinary Medical Association. San Diego, Calif. July 13, 1937. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

South Carolina Association of Veterinarians. Anderson, S. C. July 13-14, 1937. Dr. R. A. Mays, Secretary, 408-410 State Office Bldg., Columbia, S. C.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 14-15, 1937. Dr. Frank Hare, Secretary, 154 Walnut St., Lexington, Ky.

North Carolina State Veterinary Medical Association. (Joint meeting with Virginia State Veterinary Medical Association.) Wrightsville Beach, N. C. July 15-16, 1937. Dr. J. H. Brown, Secretary, Tarboro, N. C.

Virginia State Veterinary Medical Association. (Joint meeting with North Carolina State Veterinary Medical Association.) Wrightsville Beach, N. C. July 15-16, 1937. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Kansas City Veterinary Association. Auditorium, General Hospital No. 1 Annex, Kansas City, Mo. July 20, 1937. Dr. C. W. Schulz, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. July 21, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Montana Veterinary Medical Association. Great Falls, Mont. July 22-23, 1937. Dr. Hadleigh Marsh, Secretary, Agricultural Experiment Station, Bozeman, Mont.

Northwestern Ohio Veterinary Medical Association. Elks Club, Findlay, Ohio. July 27, 1937. Dr. C. W. Witty, Secretary, Elmore, Ohio.

Western Michigan Veterinary Medical Association. Ionia Fair Grounds, Ionia, Mich. July 29, 1937. Dr. Chas. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Western New York Veterinary Medical Association. Dr. F. L. Stein's farm, West Webster, N. Y. July 29, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. August 3, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. August 4, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. August 5, 1937. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

THE SIGNIFICANCE OF SUSPICIOUS AGGLUTINATION REACTIONS FOR BANG'S DISEASE*

By C. P. FITCH, LUCILLE M. BISHOP *and* W. L. BOYD
University of Minnesota Agricultural Experiment Station
Saint Paul, Minn.

When the agglutination test was first introduced as a means of diagnosis for Bang's disease of cattle, it was early recognized that reactions in the lower dilutions of the test were not always significant. Brüll,[1] M'Fadyean and Stockman,[2] Surface[3] and Seddon[4] were among the early workers to point out the fact that the serum of some apparently healthy cattle would agglutinate *Brucella abortus* organisms in the lower dilutions. Since this was found to be the case, it was necessary to determine the agglutination titres that should be considered as indicating infection. In general, it has been the policy of each laboratory to determine the agglutination titre it considered to be diagnostic. Many laboratories, however, did not formulate hard and fast rules for diagnosis but in general determined the status of the animal according to its individual history as well as that of the herd. Some of the past and present policies in regard to diagnosis will be reviewed.

Holtum[5] considered a water-clear reaction in a 1:15 dilution as probably indicative of infection. Priestley[6] stated that bovine serums showing agglutination at 1:40 were considered positive, also that agglutinins in 1:20 titres were considered positive or doubtful, depending upon whether the animal was pregnant or not. Sven Wall,[7] in a recent publication, interprets agglutination in a 1:20 dilution as signifying an infected animal. Barnes[8] considers a reaction in 1:50 as positive. Henry and Traum[9] consider any agglutination above 1:50 as positive. M'Fadyean and Stockman,[2] in 1912, considered complete agglutination in 1:50 or 1:100 as strong evidence of infection. Surface,[3] Theiler,[10] Fitch,[11] and White *et al*[12] considered agglutination in a 1:100 dilution as probably indicating infection. New York State[13] also follows this policy. Veterinary officials[14] representing state live stock sanitary authorities of California, Idaho, Nevada, Utah and Washington recommended that animals from infected herds with an agglutination titre of 1:100 be called reactors and that animals showing any trace of agglutination at a titre of 1:50

*Paper No. 1442, Journal Series, Minnesota Agricultural Experiment Station. Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

from such herds were probably showing initial infection. Smith[15] regarded a reaction in a dilution of 1:160 or above as indicating some active focus of infection in the animal, either in the uterus or the udder. Cotton and Buck[16] found that low titres (1:100 or less in tests as made by the federal Bureau) rarely indicated infection, whereas titres of 1:200 or more in over 80 per cent of the cases did.

Studies have been made in some instances in which blood agglutination titres have been correlated with bacteriological findings. Smillie, Little and Florence[17] studied 75 cases of abortions and concluded that in general a titre of 1:40 or less could be regarded as indicating that the cow was not infected. Hayes and Barger[18] state that *Brucella abortus* in the udder of a cow does not necessarily stimulate the production of blood agglutinins and that cows showing agglutinins in the blood in 1:50 dilution may frequently be udder shedders. Henry, Haring and Traum[19] state that cows with titres of 1:50 may shed *Br. abortus* at times. Schroeder and Cotton[20] tested the milk from 26 low-reacting cows (not reacting over 1:100) and found not one of them to be a carrier of *Br. abortus*. Gilman,[21] in a study of the milk of 34 cattle, was not able to isolate the organism from any animal with a blood titre lower than 1:320. Halsey,[22] in a similar study of the milk from 465 cattle, did not isolate the organism from any cow whose blood titre was not positive at 1:100 or more. Clark[23] reports isolating *Br. abortus* from the udder of one animal of 39 studied with a negative blood agglutination reaction and from three of 26 tested that gave suspicious reactions. Everson *et al*[24] studied the milk of 63 cows showing blood serum agglutination in a titre of 1:100 and found eight (12.7 per cent) positive for Brucella. Studies on the milk of 191 cows showing a blood titre under 1:100 revealed only six (3.1 per cent) positive. The blood titre of each of these animals was either on the increase or decrease at the time the sample was taken.

The above references are the bases for some of the different views and policies in regard to the diagnosis of Bang's disease by means of the agglutination test. The variations in titres considered to be diagnostic are explained in part by differences in the concentrations of the antigens. If a concentrated antigen was used, then the level of diagnosis was low; if a less concentrated antigen was used, the diagnostic level was higher. Seddon[4] and Mathews[25] were among the early workers to point out the effect of the concentration of the antigen on the agglutination titre.

The variation in the concentration of the antigen on the whole affects the actual diagnosis in only a small percentage of the

cases. All animals negative to the test may be correctly diagnosed by antigens of varied concentration. Most of the animals with high agglutination titres will be correctly diagnosed. It is particularly the animals with blood of low agglutinin content that will be materially affected.

It is evident that within almost any laboratory satisfactory results can be obtained by using the agglutination test as a means of diagnosis. However, the results of agglutination tests made in any two laboratories cannot be compared unless they are using similar antigens for performing the agglutination tests.

A step toward uniform methods of conducting and interpreting the agglutination test was made by the United States Live Stock Sanitary Association in 1931[26] and 1932.[44] It recommended a standard technic for conducting the test-tube agglutination test for Bang's disease of cattle. In addition it made certain recommendations in regard to diagnosing the disease by this method. Tests showing no reaction in the 1:25 dilution were called negative, tests showing a complete reaction in the 1:25 dilution as well as those showing an incomplete reaction in the 1:50 dilution were regarded as suspicious and tests showing reactions in 1:100 or over were called positive. The Association recognized that each herd might present an individual problem in itself and that the clinical aspects of the disease in the herd should be considered as well as complete histories of each individual animal before a diagnosis should be made.

When a herd is tested for the first time, little difficulty is encountered in the interpretation of the test if completely negative or completely positive results are obtained on the individual animal tests. The animals giving suspicious reactions are the cause of controversy and to dispose of all of them would be a costly procedure which is not always practical. It is recognized that some of these suspects may be dangerous; on the other hand, it is evident that many of them could be left in the herd and cause no spread of the disease, whereas some animals giving negative tests may react before a retest is made and be a source of infection to the herd. The initial test will give the owner of the herd some idea as to the extent of the infection, but it will not always detect all of the recently infected animals.

Statistics were collected at Minnesota from the data available under the federal testing program for Bang's disease. The agglutination titres of 30,244 cattle were recorded and it was found that 3.3 per cent of the animals gave agglutination titres within the "suspicious" range. Needless to say, not all of the 999 cattle

were condemned. Recommendations were made to isolate most of them and to treat them as suspicious animals until future tests could be made. The results of these tests confirmed them as positive or negative animals. A low percentage only remained suspicious on the second test.

The second tests of 1,074 such animals tested in Minnesota were studied, and it was found that only 204 (18.9 per cent) became positive, 806 (75.5 per cent) became negative, and 64 (5.9 per cent) remained suspicious.

The above statistics indicate that the suspicious group of animals may react in low dilutions because of an ascending titre or because of a descending titre. Some animals react in low dilution *only* and *never* have a higher agglutination titre. These three types of reactions will be discussed at length.

Reactions in Low Dilutions Due to Ascending Titres

At first glance it is obvious that certain of the animals in infected herds reacting in low dilution are dangerous animals, as the reaction may be due to a recent infection, and agglutinins have not developed in a diagnostic level. These animals may abort before a second test and spread the infection in the herd. If it were possible to make frequent retests, a greater number of infections could be detected in the earlier stages, and a quicker cleanup of a herd could be made. However, the period in the usual field experience before the first retest varies from 30 days to six months.

The period of time between the actual infection of an animal with *Br. abortus* and the appearance of agglutinins in diagnostic levels is variable depending upon the channel of infection, the amount of exposure and perhaps the stage of gestation. Birch[27] states that the average length of this period is probably about six weeks. In another publication[28] he states that when animals are infected by feeding, most of them will react to the agglutination test between the third and the eighth week, although at times the rise in titre may be very rapid. Seddon[29] states that the rise in agglutination titres from negative to positive occurs shortly after infection, may take place at any time, and bears no relation to any period of utero-gestation. M'Fadyean and Sheather[30] found that positive agglutination tests could be obtained within from seven to 21 days after infection.

The data secured at this station from two animals artificially infected this year are typical. These were heifers in their first pregnancy, were bred in our herd, and were negative to the test-tube agglutination blood test for Bang's disease. One of these

animals was drenched with 500 cc of a saline suspension prepared from the stomach contents of a Brucella-infected fetus. Blood was collected daily for the agglutination test. This heifer became positive at 1:25, 20 days later; positive at 1:50, on the 25th day; positive at 1:100, on the 30th day; positive at 1:250, on the 42nd day; positive at 1:500, on the 43rd day, and positive at 1:1,000, on the 44th day following infection. This animal aborted in 111 days, at the 240th day of pregnancy.

The other heifer, also negative to the test-tube agglutination test, was infected by placing two drops of a milky suspension of *Br. abortus* in the conjunctival sac. This animal became positive at 1:25, on the second day; positive at 1:50, on the tenth day; positive at 1:100, on the twelfth day; positive at 1:250, on the 14th day; positive at 1:500, on the 23rd day, and positive at 1:1,000, on the 58th day following infection. She aborted on the 88th day, at the 215th day of gestation.

The determination of the incubation period has to be based largely on artificial infection experiments. In naturally infected herds animals are subjected to constant reinfection, and it is impossible to obtain the definite date of infection. Experiences with infected herds show that retests made within one or two weeks after the original test will reveal new reactors, not only among the animals giving suspicious reactions on the previous test but also among the animals giving negative tests. The practice of keeping suspicious animals from such herds in isolation until further tests are made is a wise one. A large percentage of these animals may become negative and can be returned to the herd, whereas those animals that become positive on these tests have not been a source of infection.

Reactions Due to Descending Titres

Infection of bovines with Bang's disease is not always permanent, and the percentage of positive animals in an infected herd that become nonreactors to the blood test appears to be variable, and little adequate information is available on this point. It is evident that the number of reactors becoming negative would vary according to conditions in the herd, *i.e.*, hygiene, age of animals, and so forth. The important question about such animals is whether they are still potential carriers and spreaders of the disease or whether *Br. abortus* is no longer in the body of a cow whose agglutination titre descends to the suspicious or negative range. Rettger and White[31] made a study of 252 positive animals and believe that infection in adult cattle is more or less permanent in character. Birch and Gilman[32] and Giltner[33]

believe that there is a degree of immunity or resistance developed in positive cattle whose agglutination curve descends to normal. Mathews[34] divided the reacting animals in two groups, according to the curve established by the agglutination limits of their serums. The curves for the animals of group I showed no appreciable decline over a long period. The curves for animals of group II showed a gradual decline. The elimination of *Br. abortus* at the time of parturition was confined to group I. Huddleson *et al*[35] made a study of twelve animals that had been positive and whose titres had declined. The absence or disappearance of *Br. abortus* from the bodies of these animals was confirmed by repeated examinations of material suitable for bacteriological tests. Huddleson and Smith[36] further comment on this phase of the abortion problem to the effect that all animals that react to the agglutination test, even though they do not remain infected, retain a trace of agglutinins in their blood for many years. Hadley and Welsh[37] stated that two-thirds of the cows that they infected by drenching with stomach contents of aborted fetal calves became negative within six months. Beach and Humphrey,[38] in observations made at the Wisconsin Experiment Station on 14 cows which passed through an outbreak of Bang's disease and were held for observation for two years, found eleven negative or at most suspicious during this period while three at times were low reactors. Bacteriological examinations of colostrum and uterine fluid failed to reveal *Br. abortus* with the exception of one cow in one quarter of the udder at one freshening only. In a later publication,[39] seven of the above "ceased reacting" cattle were housed and pastured with 28 animals which were secured as virgin heifers from Bang's disease-free herds. At the time of the publication of this paper, there were no reactors to the blood test and eight of the heifers and five of the "ceased reactors" had calved normally. Doyle[40] studied the distribution of *Br. abortus* in the system of carrier cows and states that there is strong circumstantial evidence that the presence of specific agglutinins for *Br. abortus* indicates that this organism is or has been very recently present in the system of the animals, also that the elimination of the organism from the body appears to be rapidly followed by the disappearance of the agglutinins in detectable amounts.

Reactions in Low Dilutions Only

Perhaps the largest number of animals giving suspicious reactions to Bang's disease have developed agglutinins in the low dilutions only and never in sufficient quantities to give a positive

reaction to the test. These agglutinins may be transitory or permanent, are not well understood, and may or may not arise as a result of contact with *Br. abortus*. Lovell,[41] in a study of these types of agglutinins for the genus *Salmonella* states that they probably do not arise as a result of an actual infection and that they can be found in increasing frequency with advancing age. Jordan[42] found that older bovines have in their blood agglutinins which are scanty or absent in young animals and which are not likely to have been generated by infection with the specific homologous bacteria. Birch[27] describes the types of infection found in clean, pregnant cows when exposed for the first time to Bang bacilli. One group, he states, consists of truly immune cows that show temporary low reactions that soon disappear. These cows do not become spreaders of the infection. Gilman and Cameron[43] tested 100 bovine serums of varying Brucella agglutinin content with 15 antigens made from organisms that do or might infect cattle and reported that the low agglutination titres in Bang's disease are not due to cross-agglutination with these organisms.

Whatever may be the cause or causes of the agglutinins in low dilutions only, the fact that a large percentage of suspects belong in this group serves to complicate the agglutination test for Bang's disease. There is little doubt that these animals are not all dangerous, but the present testing methods cannot differentiate between this type of suspect and other suspects that may later become positive or dangerous animals.

EXPERIMENTAL

A study of certain types of suspects to the agglutination test for Bang's disease has been made at the University of Minnesota. This work was started in 1929 and has continued over the intervening years. The animals used for study were selected from various herds in the state after passing two or more agglutination tests and maintaining a suspicious agglutination titre. That is, their blood agglutinated *Br. abortus* in dilutions of 1:25, 1:50 or 1:100. The antigen used for obtaining these titres and the technic of the test-tube test was according to the recommendations of the United States Live Stock Sanitary Association. Suspects whose agglutination reaction was due to an ascending titre were not included in this study.

A study has been completed on 86 such animals. Twenty-three belonged in a group of suspects that had given a series of negative tests before they gave a suspicious test and never having to our knowledge a positive agglutination titre. Twenty-three

animals were former reactors and during the course of our study had agglutination titres in the suspicious or negative range. Another group of 40 suspects were studied, whose complete agglutination histories were not known, and it was not possible to determine to which of the above groups they might belong.

All of these animals have been maintained in the University's experiment herd and nearly all have been kept at least over one calving period. Bacteriological examinations of milk, colostrum, placentas, and vaginal discharges have been made to determine if *Br. abortus* was present. The possibility of these animals spreading Bang's disease to other animals was further checked by housing with them 21 negative pregnant and nonpregnant heifers.

Three barns have been used in housing the herd, and only small paddocks have been available for outside exercise. Negative controls have been housed in each barn with suspect animals and have been allowed in the same paddocks. All animals have been isolated at the time of parturition and not returned to the herd until they were free of any vaginal discharge and rectal examination indicated normal involution of the uterus. Good stable hygiene has been practiced throughout the experiment. The herd has been bled at monthly intervals and the serum tested by the test-tube agglutination test for Bang's disease. The plate method has been used from time to time to check these results. Physical examinations have been made of all animals to determine their breeding condition as well as their general health. Complete data on each are available, as to agglutination titres, breeding history, and bacteriological examinations. All of this data on 86 animals are too voluminous for publication. The histories on five individuals, which represent typical data on the entire group of animals studied, have been selected and may be found in detail in tables I, II, III, IV and V.

Milk examinations for the presence of *Br. abortus* were made on the average of every two months during the lactation periods, and colostrum was examined after every calving. In each case the samples were collected in separate containers from each quarter of the udder. The samples then were placed at refrigerator temperatures for 24 hours in order to allow the cream to rise to the surface. At least one guinea pig was inoculated with a 4-cc sample of cream from each quarter of the udder. In most cases two guinea pigs were inoculated, making a total of eight guinea pigs used for each milk or colostrum examination.

Placentas were obtained from each calving. Small pieces of chorion and cotyledons were ground in a mortar and suspended

TABLE I—*Animal* 146. *(Complete agglutination history unknown. Purchased December* 5, 1930. *Three years old. First calf, April* 10, 1930. *Herd history:* 12-3-29, 9— 18 6+; 10-28-30, 11— 1+; 11-5-30; 5— 18 [+ I —].)

BLOOD AGGLUTINATION RESULTS		BACTERIOLOGICAL EXAMINATIONS			RECTAL EXAMINATIONS	
DATE	TITRE	MATERIAL	DATE	RESULTS	DATE	FINDINGS
12– 8–30	+ I ————	Milk	12–15–30	Neg. for *Br. a.**	12– 8–30 12–13–30	Normal. Possibly in calf Removed c.l.
			Bred, December 15, 1930.			
1– 3–31	++ I ———	Milk	1–19–31	Neg. for *Br. a.*	1– 3–31	Possibly in calf
			Bred, January 23, 1931.			
2– 2–31	++ I ———					
			Bred, February 12, 1931.			
3– 3–31	++ I ———				3– 4–31	Normal
4– 2–31	++ ————				3–24–31	Possibly in calf
5– 1–31	++ ————				4–22–31	In calf
6 –1–31	++ ————				6– 1–31	In calf
7– 1–31	++ I ———					
7–30–31	+++ ———					
9– 5–31	++ ————	Nodule from leg	9–17–31	Neg. for *Br. a.*		
10– 9–31	++ I ———				10–29–31	In calf
11– 3–31	+ I ————					
			Normal calving, November 13, 1931.			
		Necrotic areas in placenta	11–13–31	Neg. for *Br. a.* Fungus present		

		placenta		Fungus present		

TABLE I—*Animal* 146. *(Complete agglutination history unknown. Purchased December* 5, 1930. *Three years old. First calf, April* 10, 1930. *Herd history:* 12-3-29, 9— 18 6+; 10-28-30, 11— 1+; 11-5-30, 5— 18 [+*I*—].)—*Continued.*

BLOOD AGGLUTINATION RESULTS		BACTERIOLOGICAL EXAMINATIONS			RECTAL EXAMINATIONS	
DATE	TITRE	MATERIAL	DATE	RESULTS	DATE	FINDINGS
		Colostrum	11–13–31	Neg. for *Br. a.*		
		Vaginal discharge	11–17–31	Neg. for *Br. a.*		
		Vaginal discharge	11–18–31	Neg. for *Br. a.*		
12– 2–31	+ I – – – – –				12– 5–31	Normal
12–31–31	+ I – – – –	Milk	1–16–32	Neg. for *Br. a.*		
2– 1–32	+ + – – – – –				2–11–32	No c. l. in either ovary
3– 1–32	+ + – – – – –	Milk	3–11–32	Neg. for *Br. a.*	3–21–32	C. l. from l. o.
		Bred, March 24, 1932.				
4– 1–32	+ + – – – – –				4–11–32	Normal
		Bred, April 14, 1932.				
					4–27–32	C. l. in r. o. Normal
5– 3–32	+ I – – – –	Milk	5–13–32	Neg. for *Br. a.*	5–17–32	In calf
6– 1–32	+ + – – – – –	Milk	6–22–32	Neg. for *Br. a.*	6– 2–32	Possibly in calf. C. l. in r. o.
7– 1–32	+ – – – – – –	Milk	7–23–32	Neg. for *Br. a.*	7–14–32	In calf
8– 1–32	+ I – – – –				8– 3–32	In calf
9– 1–32	+ – – – – – –	Milk	9–29–32	Neg. for *Br. a.*	9–28–32	In calf
10– 1–32	+ I – – – –				10–27–32	In calf
11– 1–32	+ + – – – – –				11–15–32	In calf
12– 1–32	+ – – – – – –				12– 6–32	In calf
1– 3–33	+ I – – – –				1– 5–33	In calf

TABLE I—*Animal* 146. *(Complete agglutination history unknown. Purchased December* 5, 1930. *Three years old. First calf, April* 10, 1930. *Herd history:* 12-3-29, 9— 1*S* 6+; 10-28-30,11— 1+; 11-5-30, 5— 1*S* [+*I*—].)—*Concluded.*

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
		Calved normally, January 15, 1933.				
2–15–33	+ I – – – –	Placenta Colostrum Milk	1–16–33 1–16–33 2–28–33	Neg. for *Br. a.* Neg. for *Br. a.* Neg. for *Br. a.*		
		Bred, March 1, 1933.				
3– 6–33	+ + – – – –				3– 4–33	Normal
4– 1–33	+ – – – – –				4–19–33	C. l. in l. o.
5– 5–33	+ I – – – –	Milk	5– 6–33	Neg. for *Br. a.*	5– 8–33	In calf
6– 1–33	+ I – – – –				6–31–33	In calf
7– 3–33	+ I – – – –					
		Sold, July 13, 1933.				

**Key*: Neg. = negative; *Br. a.* = *Brucella abortus;* c.l. = Corpus luteum; l. o. = left ovary; r.o. = right ovary.

TABLE II—*Animal* 280. (*Agglutination titre never over* 1:100. *Grade Holstein. Approximately three years old. Purchased December* 21, 1933, *from a mildly infected herd. Agglutination tests as follows: Dec.* 5, 1930, *April* 23, 1931, *Oct.* 22, 1931, *Feb.* 3, 1932, *May* 4, 1932, *Aug.* 24, 1932, *Dec.* 7, 1932, *March* 7, 1933, *April* 25, 1933, *June* 2, 1933, *July* 13, 1933, *Aug.* 10, 1933, *Aug.* 30, 1933 — *Negative. Sept.* 26, 1933, ++I———, *Oct.* 13, 1933, +++———, *Nov.* 16, 1933, +++———. *Herd history:* 8-10-33, 84— 18 1+; 8-30-33, 88— 98 3+; 9-27-33, 102— 118 2+; 11-17-33, 144— 148 2+.)

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
12–21–33	++I———	Milk	12–22–33	Neg. for *Br. a.**	12–27–33	In calf
1– 2–34	++———				1–31–34	In calf
2– 1–34	+———					
3– 1–34	+———	Milk	3–16–34	Neg. for *Br. a.*	2–28–34	In calf
4– 2–34	+———	Milk	4–19–34	Neg. for *Br. a.*	4– 3–34	In calf
5– 1–34	———				5– 3–34	In calf
6– 1–34	———				6– 2–34	In calf
					6–26–34	In calf
Calved normally, June 28, 1934 (partly retained afterbirth).						
7– 2–34	———	Colostrum	6–29–34	Neg. for *Br. a.*		
8– 1–34	———	Milk	7–30–34	Neg. for *Br. a.*	8–13–34	Normal
9– 1–34	———				9– 1–34	C. l. in r. o. Normal
Bred, September 8, 1934.						
10– 1–34	———	Milk	10–10–34	Neg. for *Br. a.*	10– 2–34	Small c. l. in r. o. Normal
11– 1–34	———	Milk	11–13–34	Neg. for *Br. a.*	11– 1–34	C. l. in r. o. Possibly in calf
12– 1–34	———				12– 4–34	In calf
		Milk	12–18–34	Neg. for *Br. a.*	12–18–34	In calf
Sold at South Saint Paul, December 19, 1934.						

*For key to abbreviations, see table I.

TABLE III—*Animal 272. (Complete agglutination history unknown. Purchased October 30, 1933. Four years old. Grade Holstein. Agglutination tests as follows: June 24, 1933, Sept. 13, 1933, ++I———, Oct. 25, 1933, ++———. Herd history: 6-24-33, 29— 48 9+; 10-14-33, 3— 38.)*

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
11- 2-33	++———				11- 6-33	In calf
11-28-33	+I———				12-27-33	In calf
1- 2-34	++I———					
			Calved normally, January 24, 1934.			
		Placenta	1-24-34	Neg. for *Br. a.**		
		Colostrum	1-24-34	Neg. for *Br. a.*	1-31-34	Progressing normally
2- 1-34	+++———				2-28-34	Normal
3- 3-34	++I———	Milk	3- 3-34	Neg. for *Br. a.*		
4- 2-34	++———				4- 3-34	No c. l. in either ovary
			Bred, April 2, 1934.			
5- 1-34	++———	Milk	5-25-34	Neg. for *Br. a.*	5- 3-34	C. l. in r. o. Possibly in calf
6- 1-34	++I———				6- 2-34	In calf
7- 2-34	++———				6-26-34	In calf
8- 1-34	++———	Milk	8- 3-34	Neg. for *Br. a.*	8-13-34	In calf
9- 1-34	++———				9- 1-34	In calf
10- 1-34	+++———				10- 2-34	In calf
11- 1-34	++———				11- 1-34	In calf
12- 1-34	++———				12- 4-34	In calf
1- 2-35	++I———				12-18-34	In calf
			Calved, normally, January 7, 1935.			
		Placenta	1- 8-35	Neg. for *Br. a.*		
		Colostrum	1- 8-35	Neg. for *Br. a.*		
2- 4-35	++I———	Milk	2- 8-35	Neg. for *Br. a.*	2-18-35	Calf in [illegible]

		Placenta	1– 8–35	Neg. for *Br. a.*		
		Colostrum	1– 8–35	Neg. for *Br. a.*		
2– 4–35	++I ———	Milk	2– 8–35	Neg. for *Br. a.*	2–18–35	Calving. ++ ——— ——. Retained

Holstein. Agglutination tests as follows: June 24, 1933, Sept. 13, 1933, ++I ———; Oct. 26, 1933, ++ ——— ———. Retained history: 6-24-33, 29— 4*S* 9+; 10-14-33, 3— 3*S*.)—*Concluded.*

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
		Bred, February 28, 1935.				
3– 4–35	++———					
		Bred, March 26, 1935.				
4– 1–35	++———	Milk	4–27–35	Neg. for *Br. a.*	4–13–35	C. l. in l. o.
					4–26–35	Endometritis. No c. l. in either ovary
5– 1–35	++———				5–22–35	Hydrometra. Small c. l. in l. o.
6– 1–35	++———				6– 6–35	Cystic r. o. Cyst broken
					6–18–35	Normal. C. l. in l. o.
		Bred, June 25, 1935.				
		Milk	6–26–35	Neg. for *Br. a.*		
7– 1–35	++———				7–16–35	C. l. in r. o. Possibly in calf
8– 1–35	++———				8–21–35	In calf
8–31–35	++———	Milk	9–23–35	Neg. for *Br. a.*	9–24–35	In calf
10– 1–35	++———				10–17–35	In calf
11– 1–35	++———				11–14–35	In calf
12– 3–35	++I ———	Milk	12– 6–35	Neg. for *Br. a.*	12–14–35	In calf
1– 2–36	+I ———				1–16–36	In calf
2– 1–36	++———				2–15–36	In calf
3– 2–36	++———					
		Calved, March 3, 1936 (dead twins).				
3– 4–36	++I ———	Colostrum	3– 3–36	Neg. for *Br. a.*		
		Placenta	3– 3–36	Neg. for *Br. a.*		
3–17–36	++I ———	Twin fetuses	3– 3–36	Neg. for *Br. a.*	3–14–36	Progressing normally
		Sold at South Saint Paul, March 17, 1936.				

*For key to abbreviations, see table I.

TABLE IV—*Animal* 138. *(Complete agglutination history unknown. Purchased June* 13, 1930. *Grade Guernsey. Born February*, 1926. *Agglutination tests as follows: Oct.* 16, 1929, +——, *March* 10, 1930, +——, *May* 10, 1930, ———, *June* 3, 1930, +++———. *Herd history: Badly infected.* 11-16-29, 73— 7*S* 21+; 3-10-30, 52— 9*S* 21+.)

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
			Bred, June 18, 1930.			
7– 1–30	———————	Milk	7–17–30	Neg. for *Br. a.**	7–16–30	Possibly pregnant. C. l. in r. o.
8– 1–30	++———	Milk	8– 6–30	Neg. for *Br. a.*		
9– 2–30	+I———	Milk	9–23–30	Neg. for *Br. a.*	9– 3–30	In calf
10– 2–30	+I———	Milk	10–22–30	Neg. for *Br. a.*	9–19–30	In calf
11– 3–30	++———					
12– 2–30	———————	Milk	12–10–30	Neg. for *Br. a.*		
1– 3–31	+++———					
2– 2–31	+++———					
3– 3–31	++———					
			Aborted, March 7, 1931.			
		Placenta	3– 7–31	Yeast isolated		
		Fetus	3– 7–31	Yeast isolated		
		Colostrum	3– 7–31	Neg. for *Br. a.*		
4– 2–31	++I———					
5– 1–31	II———	Milk	5– 1–31	Neg. for *Br. a.*		
			Bred, May 13, 1931.			
6– 1–31	+———	Milk	6– 6–31	Neg. for *Br. a.*	6– 1–31	C. l. in l. o.
			Bred, June 15, 1931.			
7– 1–31	++———					

TABLE IV—*Animal* 138. *(Complete agglutination history unknown. Purchased June* 13, 1930. *Grade Guernsey. Born February*, 1926. *Agglutination tests as follows: Oct.* 16, 1929, + — —, *March* 10, 1930, + — —, *May* 10, 1930, — — —, *June* 3, 1930, + + + — — —. *Herd history: Badly infected.* 11-16-29, 73— 7*S* 21+; 3-10-30, 52— 9*S* 21+.)—*Continued.*

BLOOD AGGLUTINATION RESULTS		BACTERIOLOGICAL EXAMINATIONS			RECTAL EXAMINATIONS	
DATE	TITRE	MATERIAL	DATE	RESULTS	DATE	FINDINGS
			Bred, July 7, 1931.			
7–30–31	+ — — — — —					
9– 5–31	+ + — — — —				9– 9–31	In calf
10– 9–31	+ + — — — —				10–29–31	In calf
11– 3–31	+ — — — — —	Milk	11–25–31	Neg. for *Br. a.*		
12– 2–31	+ + + — — —				12– 5–31	In calf
12–31–31	+ I — — — —	Milk	1–16–32	Neg. for *Br. a.*	12–21–31	In calf
2– 1–32	+ + + — — —					
3– 1–32	+ + I — — —					
4– 1–32	+ + I — — —				4–12–32	In calf
			Normal calving, April 14, 1932.			
		Placenta	4–14–32	Neg. for *Br. a.*		
		Colostrum	4–14–32	Neg. for *Br. a.*		
		Vaginal discharge	4–18–32	Neg. for *Br. a.*		
		Vaginal discharge	4–21–32	Neg. for *Br. a.*		
		Vaginal discharge	4–25–32	Neg. for *Br. a.*	4–27–32	Normal discharge. Progressing normally. C. l. in l. o. very small
5– 3–32	+ + — — — —	Milk	5–17–32	Neg. for *Br. a.*	5–17–32	Normal

TABLE IV—*Animal* 138. (*Complete agglutination history unknown. Purchased June* 13, 1930. *Grade Guernsey. Born February*, 1926. *Agglutination tests as follows: Oct.* 16, 1929, + — —, *March* 10, 1930, + — —, *May* 10, 1930, — — —, *June* 3, 1930, + + + — — —. *Herd history: Badly infected.* 11-16-29, 73— 7*S* 21+; 3-10-30, 52— 9*S* 21+.)—*Concluded.*

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
			Bred, June 3, 1932.			
6– 1–32	+ I — — — —	Milk	6–22–32	Neg. for *Br. a.*	6–24–32	C. l. in r. o.
7– 1–32	+ + — — — —	Milk	7–25–32	Neg. for *Br. a.*	7–14–32	In calf
8– 1–32	+ I — — — —				8– 3–32	In calf
					8–22–32	In calf
9– 1–32	— — — — — —				9– 1–32	In calf
9–20–32	+ I — — — —	Milk	9–20–32	Neg. for *Br. a.*		

Slaughtered and autopsied, September 20, 1932. *Br. abortus* negative.

*For key to abbreviations, see table I

TABLE V—*Animal* 257. *(Agglutination titre never over* 1:100. *Purchased July* 27, 1933. *Purebred Holstein. Born October* 18, 1931. *Agglutination tests as follows: March* 17, 1932, *Aug.* 17, 1932, *Dec.* 28, 1932, *Negative. May* 2, 1933, +*I* — — — —, *June* 14, 1933, +*I* — — — —, *July* 20, 1933, +++ — — —. *Herd history: Mildly infected.* 3-19-32, 135— 3*8;* 5-3-33, 141— 1*8* 1+; 6-15-33, 11— 1*8* 1+.)

Blood Agglutination Results		Bacteriological Examinations			Rectal Examinations	
Date	Titre	Material	Date	Results	Date	Findings
7–27–33	+++ — — —					
8– 1–33	++ — — — —					
8–21–33	++ I — — —				8–21–33	In calf
9– 1–33	++ — — — —				8–24–33	In calf
10– 2–33	++ — — — —				9–25–33	In calf
11– 2–33	+ I I — — —				10–25–33	In calf
11–30–33	+ I — — — —				11–16–33	In calf
12–12–33	++ — — — —				12–27–33	In calf
1– 2–34	I I — — — —					
		Normal calving, January 3, 1934.				
		Placenta	1– 3–34	Neg. for *Br. a.*		
		Colostrum	1– 3–34	Neg. for *Br. a.*		
		Milk	1–11–34	Neg. for *Br. a.*	1–31–34	No c. l. in either ovary. Normal
2– 1–34	+ — — — — —				2–28–34	No c. l. in either ovary
3– 1–34	+ I — — — —	Milk	3–15–34	Neg. for *Br. a.*	3– 6–34	Ruptured ripe follicle in l. o. May establish estrum

TABLE V—*Animal* 257. (*Agglutination titre never over* 1:100. *Purchased July* 27, 1933. *Purebred Holstein. Born October* 18, 1931. *Agglutination tests as follows: March* 17, 1932, *Aug.* 17, 1932, *Dec.* 28, 1932, *Negative. May* 2, 1933, $+I$ — — — —, *June* 14, 1933, $+I$ — — — —, *July* 20, 1933, $+++$ — — —. *Herd history: Mildly infected.* 3-19-32, 135— 3*S*; 5-3-33, 141— 1*S* 1+; 6-15-33, 11— 1*S* 1+.)—*Concluded.*

BLOOD AGGLUTINATION RESULTS		BACTERIOLOGICAL EXAMINATIONS			RECTAL EXAMINATIONS	
DATE	TITRE	MATERIAL	DATE	RESULTS	DATE	FINDINGS
			Bred, March 18, 1934.			
4– 2–34	+ — — — — —				4– 3–34	C. l. in l. o.
5– 1–34	+ I — — — —				5– 3–34	Possibly in calf
			Bred, May 4, 1934.			
6– 1–34	+ + — — — —	Milk	5–21–34	Neg. for *Br. a.*	6– 2–34	Possibly in calf
			Bred, June 12, 1934.			
					6–26–34	C. l. in r. o. Normal
7– 2–34	+ + I — — —				7–17–34	In calf
8– 1–34	+ I — — — —	Milk	8– 6–34	Neg. for *Br. a.*	8–13–34	In calf
9– 1–34	+ + I — — —				9– 1–34	In calf
			Slaughtered and autopsied, September 25, 1934. *Br. abortus* negative.			

*For key to abbreviations, see table I.

in saline. Cultures were made on gentian violet plates (gentian violet 1:200,000) and intraperitoneal inoculations also were made into three guinea pigs with this saline extract.

If abortions occurred or if calves died shortly after birth, cultures were made of the lung, liver, spleen, and stomach contents, and two guinea pigs were inoculated with 3 cc of the stomach contents and two were also inoculated with 2 cc of a saline extract of lung tissue.

In a few cases placental tissue was not obtainable for culture or guinea pig inoculations and in these cases vaginal swabs were made. The swabs were soaked in saline and guinea pig inoculations were made with the saline suspension. In some cases vaginal swabs were made every three or four days for a period of two weeks after calving.

Complete autopsies were made of ten animals and bacteriological examinations were made of lymph-glands, uterus, udder, spleen and liver. These examinations consisted of direct cultures and guinea pig inoculations. In addition, the udder and supramammary lymph-glands were obtained for study from one animal, and the uterus was obtained from another animal.

All cultures of the above materials were made on serum agar. If the material was contaminated, such as placental tissues, cultures were also made on serum agar containing gentian violet in a 1:200,000 dilution. All cultures were incubated in an atmosphere of 10 per cent carbon dioxide for four to six days before they were examined for growth. At this time all suspicious colonies were subcultured and the subcultures were examined to determine if they were *Br. abortus*.

All guinea pigs inoculated with milk or tissues were autopsied four to five weeks after inoculation. In each case the agglutination titre of the blood serum was determined and cultures were made of the spleen.

The technic described for direct culture of *Br. abortus* as well as the guinea pig inoculation method has been very satisfactory for the isolation of *Br. abortus* from cows with agglutination titres of over 1:100. Tables VI and VII show the number of examinations made as well as the lactation periods covered.

Results

1. Three individuals from the suspicious groups of animals have become positive to the agglutination test during the course of the experiment. Two of them aborted and *Br. abortus* was isolated from the placenta and colostrum. Two had been pre-

For key to abbreviations, see table I.

STATE UNIV

vious reactors, that is, with titres above 1:100. The other individual gave a maximum titre of an incomplete at 1:100 approximately one year before she showed a higher titre and aborted. All three gave definite evidence by the blood test of infection in ample time to be removed from the herd before any of them aborted.

2. *Br. abortus* was not isolated by any bacteriological examination made of suspicious animals in the herd. None of the

TABLE VI—*Number of examinations made for Brucella abortus.*

ANIMALS		PLACENTA AND COLOSTRUM	MILK	FETUSES AND CALVES		VAGINAL DISCHARGES	AUTOPSIES
CLASSIFICATION	NUMBER						
Former reactors	23	34	156	Fetuses 7-day calf Premature twins	3 1 2	37	6
Agglutination titres never over 1:100	23	31	134	Fetuses	5	6	2
Agglutination histories unknown	40	55	275	Fetuses 7-day calf Premature twins	6 1 4	50	4
Control animals	21	35	114	Fetuses	5	2	0
Totals	107	155	679		27	95	12

TABLE VII—*Number of calving periods studied.*

	ANIMALS NOT CALVING	THROUGH ONE CALVING PERIOD	THROUGH TWO CALVING PERIODS	THROUGH THREE CALVING PERIODS	THROUGH FOUR CALVING PERIODS
Former reactors	3	11	7	1	1
Animal agglutination titres never over 1:100	1	15	5	2	
Animal agglutination histories unknown	4	21	11	4	
Control animals	1	9	7	4	
Totals	9	56	30	11	1

control animals have become infected although some of them have developed titres of 1:50 and at times 1:100.

Discussion

This study has shown that one is able, through careful selection and study, to arrive at a reasonably accurate diagnosis and disposition of particular individuals showing suspicious reactions to Bang's disease. A definite rule for the disposition of all animals having suspicious titres for Bang's disease on a single test is impossible. It is necessary to have the results of a series of tests over a considerable period of time and information on the duration of pregnancy along with an adequate herd history in order that the proper deductions can be made. The results so far obtained show conclusively that most animals having a constant suspicious titre for Bang's disease are not dangerous and will not spread Bang infection in an otherwise clean herd. This study is being continued and more information will be available.

References

[1]Brüll, Z.: Beitrag zur Diagnostik des infektiösen Abortus des Rindes. Berl. Tierärzt. Wchnschr., xxvii (1911), p. 721.

[2]M'Fadyean, J., and Stockman, S.: The agglutination test in the diagnosis of bovine contagious abortion. Jour. Comp. Path. & Therap., xxv (1912), pp. 22-29.

[3]Surface, F. M.: The diagnosis of infectious abortion in cattle. Ky. Agr. Exp. Sta. Bul. 166 (1912).

[4]Seddon, H. R.: Some observations on the methods of using the agglutination test in the diagnosis of the disease in bovines caused by the bacillus of contagious abortion. Jour. Comp. Path. & Therap., xxviii (1915), pp. 20-36.

[5]Holtum, A. W.: A determination of the non-specific agglutinin content of normal bovine sera, and its bearing on the interpretation of the agglutination test for bovine contagious abortion. Jour. Comp. Path. & Therap., xlii (1929), pt. 1, pp. 33-39.

[6]Priestley, F. W.: The incidence in Great Britain of *Br. abortus* infection in various domestic animals as indicated by the agglutination test. Jour. Comp. Path. & Therap., xlvii (1934), pt. 3, pp. 181-189.

[7]Wall, S.: Om agglutinationsvärdet 10 och dess betydelse för serologisk diagnos av sjukdomen smittsam kastning hos nötkreatur. Skand. Vet.-Tids., xxvi (1936), pp. 413-455.

[8]Barnes, M. F.: Bang's disease. Corn. Vet., xx (1930), pp. 249-262.

[9]Henry, B. S., and Traum, J.: A comparison of factors influencing the agglutination test for *Brucella abortus*. Jour. Inf. Dis., xlvii (1930), pp. 367-379.

[10]Theiler, A.: Contagious abortion in cattle. Rhodesia Agr. Jour., xv (1918), pp. 268-278.

[11]Fitch, C. P.: Diagnosis of bovine contagious abortion. Jour. A. V. M. A., lvi (1920), n. s. 9 (5), pp. 459-467.

[12]White, G. C., Johnson, R. E., Plastridge, W. N., and Reece, R. P.: A study of agglutination reactions in the diagnosis of Bang's disease. Storrs Agr. Exp. Sta. Bul. 185 (1933).

[13]Gilman, H. L.: What should constitute a satisfactory Bang's disease test for interstate shipment? Vet. Med., xxviii (1933), pp. 37-38.

[14]——— Recommended standard for *Brucella abortus* agglutination test certificates in Pacific Coast and Rocky Mountain states. Corn. Vet., xix (1929), pp. 251-252.

[15]Smith, T.: Remarks on the etiology of infectious abortion in cattle. Corn. Vet., xi (1921), pp. 85-91.

[16]Cotton, W. E., and Buck, J. M.: Bureau of Animal Industry researches on infectious abortion. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (3), pp. 306-325.

[17]Smillie, E. W., Little, R. B., and Florence, L.: An interpretation of the agglutination reaction to *Bacillus abortus* in 75 cases of bovine abortion bacteriologically controlled. Jour. Exp. Med., xxx (1919), pp. 341-357.

[18]Hayes, F. M., and Barger, E. H.: Bang's disease in a naturally infected herd. Hilgardia, ix (1935), pp. 527-542.
[19]Henry, B. S., Haring, C. M., and Traum, J.: *Brucella abortus* shedder conditions in twenty cows. Hilgardia, ix (1935), pp. 545-566.
[20]Schroeder, E. C., and Cotton, W. E.: Carriers of Bang abortion bacilli and the agglutination test. Jour. A. V. M. A., lxiv (1924), n. s. 17 (4), pp. 479-481.
[21]Gilman, H. L.: The elimination of *Bact. abortus* in the milk of cows. Corn. Vet., xx (1930), pp. 106-122.
[22]Hasley, D. E.: Further studies on *Brucella abortus* in certified milk. Amer. Jour. Pub. Health, xxi (1931), pp. 515-518.
[23]Clark, C. F.: Correlation of blood reactions with the breeding records over a period of years in an abortion-infected herd of cattle. Jour. A. V. M. A., lxxix (1931), n. s. 32 (3), pp. 290-331.
[24]Everson, C. L., Poelma, L. J., and Brueckner, A. L.: The relation of blood and milk sera agglutination titres to udder infection in Bang's disease. Md. Agr. Exp. Sta. Bul. 387 (1935), pt. 2, pp. 395-400.
[25]Mathews, F. P.: A study of the agglutination test for bovine infectious abortion. Jour. Inf. Dis., xxxv (1924), pp. 498-501.
[26]Report of Committee on Bang's Disease of the U. S. Live Stock Sanitary Association for 1931 (pt. 1). Jour. A. V. M. A., lxxx (1932), n. s. 33 (3), pp. 323-326.
[27]Birch, R. R.: Some principles underlying official regulations for the control of Bang's disease. Corn. Vet., xxii (1932), pp. 134-140.
[28]Birch, R. R.: The nature and economic importance of Bang's disease of cattle. An. Rpt. N. Y. State Vet. Coll., Cornell Univ., 1931-32 (1933), pp. 163-170.
[29]Seddon, H. R.: Studies in abortion disease. Jour. Comp. Path. & Therap., xxxii (1919), pp. 1-34.
[30]M'Fadyean, J., Sheather, A. L., and Minett, F. C.: Researches regarding epizoötic abortion of cattle. Jour. Comp. Path. & Therap., xxvi (1913), pp. 142-170.
[31]Rettger, L. F., and White, G. C.: Infectious abortion in cattle. Storrs Agr. Exp. Sta. Bul. 93 (1918), pp. 199-248.
[32]Birch, R. R., and Gilman, H. L.: The agglutination test as an aid in handling Bang abortion disease. Corn. Vet., xvi (1926), pp. 127-132.
[33]Giltner, W.: The present status of infectious abortion control from the laboratory standpoint. Jour. A. V. M. A., lxxiv (1929), n. s. 27 (6), pp. 880-884.
[34]Mathews, F. P.: The diagnosis of infectious abortion with special reference to agglutinating limits. Jour. A. V. M. A., lxix (1926), n. s. 22 (5), pp. 613-621.
[35]Huddleson, I. F., Torrey, J. P., and Carlson, E. R.: The relation of the agglutination test to the presence of *Brucella abortus* in the body of the bovine. Jour. A. V. M. A., lxxii (1928), n. s. 25 (6), pp. 889-901.
[36]Huddleson, I. F., and Smith, L. H.: A critical study of the Brucella agglutination reaction and abortion rate in a herd of cattle under natural conditions. Jour. A. V. M. A., lxxix (1931), n. s. 32 (1), pp. 63-78.
[37]Hadley, F. B., and Welsh, W. E.: On the persistency of the agglutination reaction for *Brucella abortus*. Corn. Vet., xxi (1931), pp. 286-291.
[38]Beach, B. A., and Humphrey, G. C.: The presence of *Br. abortus* in the uterine fluid and in the milk and of agglutinins in the blood sera of so-called ceased reactor cows. Vet. Med., xxx (1935), pp. 8-10.
[39]——— Can ceased reactors to the Bang's test safely be allowed to mingle with susceptible animals? An. Rpt. Dir. Wis. Agr. Exp. Sta. Bul. 435 (1936), pp. 21-22.
[40]Doyle, T. M.: The distribution of *Br. abortus* in the system of "carrier" cows. Jour. Comp. Path. & Therap., xlviii (1935), pt. 3, pp. 192-217.
[41]Lovell, R.: The presence of agglutinins for bacteria of the Salmonella group in the sera of normal animals. Jour. Comp. Path. & Therap., xlv (1932), pp. 27-42.
[42]Jordan, E. O.: Influence of age on amount of normal agglutinins in the blood of cattle. Proc. Soc. Exp. Biol. & Med., xxx (1933), pp. 446-447.
[43]Gilman, H. L., and Cameron, H. S.: The significance of low agglutination titers in Bang's disease. Corn. Vet., xxvi (1936), pp. 113-119.
[44]Report of Committee on Bang's Disease of the U. S. Live Stock Sanitary Association for 1932 (pt. 2). Jour. A. V. M. A., lxxxii (1933), n. s. 35 (3), pp. 335-337.

Discussion

Dr. R. R. Birch: Dr. Fitch made one statement for which I would like to know just what the basis is. He made the statement "that once a reactor always a suspicious animal." Does he apply that to young heifers?

Dr. Fitch: I did not make that statement. I made the statement, "Once a reactor always dangerous," and it applies only to adult cows.

DR. BIRCH: It applies only to adult cows. That is, if you followed the record of an adult cow for perhaps a year, which had a reaction previously, and you continued with tests which were all negative, you would still hold the cow under suspicion? What are the supporting data for that statement, Doctor?

DR. FITCH: We had 10 per cent of our cows which were at first positive, went negative and aborted later.

DR. BIRCH: Did they react?

DR. FITCH: Yes.

DR. BIRCH: Then the comparison between those cows and the cows which began with a negative test should be made. Whether that is a case of a reinfection, or whether it is a case of a hangover is something which should be distinguished.

DR. FITCH: You cannot say there is no opportunity for a reinfection. I do not know how you could do that.

DR. BIRCH: I do not think you can.

DR. FITCH: I do not think you can. I want that clearly understood; but as far as we could make it out, there was no opportunity for reinfection.

DR. BIRCH: We have followed a great many of those cows' records, both in the field and in experiment herds, just as you did, and we have been doing it for a period of about twelve years. We made tests on these individual cows each month, examined their milk, and the placenta at each calving, regardless of whether it was normal or abnormal. We reached essentially the same conclusion with respect to the danger. That is, the danger in those cows which maintained a low titre seemed to be at a minimum. I think I would want to see more evidence before I reached the conclusion that a cow which will maintain a low titre, which has been up, is any more likely to develop a titre subsequently than the negative cow. We simply do not find evidence to that effect.

DR. B. A. BEACH: I would like to report briefly our experience along the same line.

Fourteen so-called "cease reactors" were placed in a negative herd; that is, a group of negative heifers were placed with these 14 cease reactors. They were cease reactors from time to time; at other periods, they were not entirely so. We followed their records for a period of something over two years. One of the heifers among the negative group placed in this group developed a titre of 1:100, was taken out, calved normally, and we were unable to locate *Br. abortus* in that heifer anywhere. Whether she was infected or not, I do not know. She reacted in 1:100, but we failed to uncover the organism.

DR. BIRCH: What about the others? Did you make an examination of the others to see whether they were infected?

DR. BEACH: Yes. We did that.

DR. BIRCH: Did you find anything?

DR. BEACH: We did not. That was done at every calving period on all the cows, the old ones and the heifers in the herd.

There was one exception to that. There was one cow which was negative, and had been entirely negative except for a very slight indication to the contrary on some tests for a period of possibly a year or a year and a half; and we were testing them every two weeks. Following one calving period, we did pick up Brucella in one quarter, unmistakably. She remained negative to the end of her days; and this was not long for she died in an accident. We failed to find *Br. abortus* at the time she was slaughtered. I do not know if she was responsible for the infection of this one heifer, if she was infected.

That left 13; twelve of those were infected just before the experiment was concluded. One cow was not infected because she was not pregnant at the time, and she was taken out of the group. The others

were 60 days pregnant, or as near to 60 days as could allow for the whole herd. Eleven of the twelve did not experience a rise in the titre even after infection. They carried to full term. In other words, as shown by this test, eleven of them were immune. The twelfth one was highly susceptible. I do not know why. She reacted very promptly after infection and aborted, and *Br. abortus* was isolated from the expelled material.

The history of that cow is interesting. This herd had been infected approximately five years before. This cow, while we called her a "cease reactor," was not one; she never reacted. At the time of the initial infection she did not react. She carried a half to full term, and we never located the organisms in that cow. She is what is known as an "escape." Why they escape, I do not know, but according to the experience on this one herd—and it was subsequently confirmed to quite an extent on another herd—the cows which ceased to react, or nearly so, are, in the majority of cases, immune thereafter, or for a period of years. We cannot, of course, draw definite conclusions because our numbers are too small.

DR. BIRCH: I know in following those cases we do get an occasional animal which is reinfected; but we know also that the vast majority of them are resistant and we do not find them acting as spreaders; that is, eliminating those cases in which a definite reaction appears. I am speaking now of those who do maintain the low titres. We look at them just the same as we do a negative animal, because we know that in our initial tests if we go into any herd there are likely to be some of those animals. They are left in the herd. As a matter of fact, we do not know which ones they are.

DR. H. L. GILMAN: I am a little hazy in my own mind about one point which Dr. Fitch mentioned. Did you advocate the segregation of suspicious animals in the herd until subsequent tests have given a definite determination of the trend of the reaction?

DR. FITCH: No. These animals were purchased as suspicious animals only after at least two agglutination tests, at which they had maintained a suspicious titre, had been made. They were placed in the main herd then and kept there during the period of the experiment.

DR. F. B. GRAVES: Would you consider a suspicious cow at 1:50 safe in a raw milk supply?

DR. FITCH: I would if she maintained it as a constant titre.

DR. J. D. RAY: I would like to ask Dr. Fitch if he would expect to continue to get reactors in a herd if he had a number of 1:50 reactors and left them in the group; whether, if he considers that a clean herd, he would expect to keep getting positive reactors from that herd?

DR. FITCH: No, sir. I answer that question on the basis that we have tabulated over 1,000 of those herds in which we have left that type of animal in the groups. We have tabulated on the second test and we do not get any appreciable number of reactors from those herds.

DR. BIRCH: We can confirm that statement as being the general experience. With the 1:50 reactor, with no other higher reactors in the herd, we usually find in a large herd that an animal perhaps has gone down, and in another one it is up, but that seems to be what we might term the limit of the normal upward swing. That is not very well expressed, but I do not know how else to state it.

DR. BEACH: As Doctor Birch has said, the titre of this particular class of cows will not remain constant. They will go up and down within certain limits; and the limited experience we have had confirms both Dr. Fitch's and Dr. Birch's statements. The only dangerous cow in our herd was one that did not show any titre whatsoever; and some which were 1:50 were not dangerous.

DR. A. A. HUSMAN: Dr. Fitch, if you were setting up some rules for accreditation of Bang's disease-free herds and you had in a number of these herds animals which reacted consistently at 1:25, or even 1:50, after how long a period would you think it would be safe to set the herds up as Bang's disease-free?

DR. FITCH: I can answer that question by saying that we formulate no general rule on that subject. It depends upon the type of herd in which those animals occur. If they are negative in the beginning, two or three negative tests are ample for accrediting that herd.

Veterinary Practice Acts Changed

The Massachusetts Veterinary Practice Act has been amended to provide that an applicant for a license to practice must be a citizen of the United States in order to be eligible to take the state board examinations. The new law has been signed by the Governor and a photostatic copy forwarded to the Massachusetts Veterinary Association.

In Kansas, Senate Bill 25 became a law on March 30, 1937. It increases the annual registration fee for veterinarians from two dollars to four dollars. Considerable difficulty had been encountered in enforcing the veterinary practice act with the limited funds provided by the old registration fee. The new fee is expected to yield at least $1,000 more than the old one, and will make possible the thorough investigation of violations of the law and the prosecution of offenders.

Minnesota has a new practice act effective May 1. It contains a number of features which are believed to be distinct improvements over the old law, particularly in connection with the prosecution of violators of the act. It is to be regretted that the new law specifically exempts the treatment of poultry and birds, but it is reported that the Minnesota veterinarians had to compromise with the hatchery interests in order to secure passage of the new legislation. Whether this was a wise move remains to be seen.

Northwest Indiana Veterinarians Organize

Eighteen veterinarians from Jasper and surrounding counties met in Rensselaer, Ind., on June 10, 1937, and organized the Northwest Indiana Veterinary Association. Dr. G. W. Andree, of Rensselaer, acted as host. A six o'clock dinner was served at the Lowman House and officers of the new organization were selected as follows: President, Dr. J. E. Tinder, of Brook, Ind., and secretary-treasurer, Dr. L. E. Andres, of Remington, Ind.

SALMONELLA INFECTIONS IN CHICKENS*

By H. VAN ROEKEL *and* K. L. BULLIS, *Amherst, Mass.*
Department of Veterinary Science
Massachusetts Agricultural Experiment Station

Within recent years, considerable interest has been manifested in diseases whose etiogolical agents represent members of the *Salmonella* genus. The losses occurring from Salmonelloses in chickens have mounted to great proportions, due to the fact that the members of the *Salmonella* genus (some more than others) are not only widespread but also very greatly concentrated in certain geographical sections. Within the last ten years, an unusually rapid development has been made in incubation and hatching operations for the poultry industry. These developments, along with modern traffic facilities, have exerted a great influence on the dissemination and prevalence of Salmonella diseases.

Salmonelloses in chickens may involve the following organisms: *S. pullorum, S. gallinarum, S. aertrycke, S. anatum* and possibly others in the *Salmonella* genus. Among the organisms mentioned, *S. pullorum* appears to be the most widely disseminated in this country. Since pullorum disease is recognized as one of the leading poultry diseases in respect to economic losses to the industry, the greater part of this paper is devoted to *S. pullorum* infection.

SIGNIFICANCE OF CHARCTERISTICS OF S. PULLORUM

Researches concerning pullorum disease have accumulated knowledge to the point where methods of control and eradication of the disease appear to be the remaining important problems. However, those engaged in combating pullorum disease should not overlook certain phases of this disease which may have an indirect or a direct influence on methods of control and eradication.

The findings of Plasteridge and Rettger,[1] Mallmann,[2] and Van Roekel and Rettger[3] have revealed that *S. pullorum* possesses potentialities for atypical characters. Such potential variation from the so-called normal may occur under natural conditions both in the farm flock and in the laboratory without any pre-arranged effort to bring about atypical behavior of the organism. To what extent *S. pullorum* variation is operative in farm poultry flocks remains a question. In testing for the pullorum-disease "carrier" it is possible that variation of the organism may be responsible for such types of reactions as "doubtful," "intermit-

*Contribution No. 249 of the Massachusetts Agricultural Experiment Station. Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

tent," and "non-specific." Variation of the organism may also manifest itself in antigens employed for the macroscopic agglutination test. In the preparation of a test fluid for the agglutination test it is essential that stable, typical *S. pullorum* strains be employed. Likewise, hyposensitive or hypersensitive strains should not be used in antigen for routine testing. Stock antigen strains should be maintained under a constant suitable environment and checked at frequent intervals for characteristic properties of *S. pullorum*.

Control and Eradication

While pullorum disease is recognized as a disease producing its greatest losses among baby chicks, it occasionally may give rise to heavy mortalities in growing and adult birds. Furthermore, the fact that the disease is carried along in adult birds which in turn may transmit the organism to their progeny by means of the egg, affords a vulnerable point of attack by those combating the disease. Much effort has been exerted in the control of the disease among baby chicks by such means as fumigation of incubators, rigid culling, modifying brooder management, medication of drinking water, ultra sanitation practices, and isolation methods. Although such methods of control may have exerted a temporary beneficial influence, they cannot be expected to eliminate losses from pullorum disease completely. This fact has long been appreciated in certain sections where eradication of the disease from flocks has been adopted as the goal.

The macroscopic agglutination test has played a very important part in the eradication of the disease from flocks. However, it should be recognized as only one of several important steps in the eradication of the disease. An effective disease elimination program can be established and maintained only on sound eradication principles. This fact is not appreciated by some who have taken the reins of pullorum-disease testing into their hands.

In the early stages of pullorum-disease testing, considerable skepticism existed as to its effectiveness in eliminating infected birds from the flock. This sentiment existed in Massachusetts when the work was first undertaken. However, through the improvement of testing methods, adoption of effective eradication principles, and accumulated testing results, poultrymen have come to realize the value of pullorum-disease eradication.

In table I is given a ten-year testing summary of Massachusetts flocks. The data show a gradual reduction in the annual per-

centages of positive tests, a ten-year decrease from 4.03 per cent to 0.30 per cent. The percentages of non-reacting flocks of the total tested for the different years show a gradual increase during the ten-year period. In 1927, 45.78 per cent of the total tested flocks were non-reacting, while in 1936, the percentage had mounted to 91.27. The data concerning the percentage of total birds in non-reacting flocks are of still greater interest. In 1927, 31.63 per cent of the birds were in non-reacting flocks and during the ten-year period the percentage had increased to 95.95. The average flock size was smaller in 1927 than in 1936.

TABLE I—*Ten-year testing summary of Massachusetts flocks.*

TESTING SEASON	FLOCKS	TOTAL TESTS	POSITIVE TESTS (%)	TESTED FLOCKS NON-REACTING (%)	BIRDS IN NON-REACTING FLOCKS (%)
1926–27	249	127,327	4.03	45.78	31.63
1927–28	321	190,658	6.52*	42.99	42.40
1928–29	413	254,512	4.25	55.21	60.25
1929–30	460	386,098	2.17	67.17	66.97
1930–31	447	402,983	1.47	73.38	74.89
1931–32	455	420,861	0.90	78.02	79.15
1932–33	335	300,714	0.47	82.39	80.41
1933–34	262	284,848	0.53	87.40	80.83
1934–35	244	301,887	0.39	87.30	89.56
1935–36	252	344,081	0.30	91.27	95.95

*Based on total birds tested.

In table II are presented data of flocks tested for the first time and flocks tested for three or more consecutive years. It is quite evident that the percentages of positive tests throughout the eight-year period are markedly less for the flocks tested three or more consecutive years. Also, the percentages of non-reacting flocks are greater in the three-year group. Unquestionably annual testing of all the birds on the premises has been effective in eliminating infection from individual flocks and reducing pullorum disease among the flocks as a whole. First-year tested flocks have in recent years benefited by the pullorum-clean flocks established through annual testing. This fact appears to manifest itself in the percentages given for the flocks tested for the first time.

RETESTING OF INFECTED FLOCKS

The practice of eradicating pullorum infection from flocks through short-interval testing (four to six weeks), until no more reactors are revealed, has not met with general acceptance by

TABLE II—*Comparative testing data of flocks tested for the first time and flocks tested for three or more consecutive years.*

CLASSIFICATION	1928–29	1929–30	1930–31	1931–32	1932–33	1933–34	1934–35	1935–36
Total flocks:								
First year	145	122	89	93	56	37	37	43
Three or more years	121	199	253	269	219	178	161	151
Total tests:								
First year	44,935	45,257	36,520	29,507	17,854	14,140	22,790	21,892
Three or more years	109,093	241,581	305,716	334,699	245,617	245,672	247,087	271,410
Positive tests (%):								
First year	7.01	5.63	4.78	5.07	3.03	1.26	2.17	2.55
Three or more years	3.35	1.18	0.90	0.46	0.21	0.34	0.18	0.10
Non-reacting flocks:								
First year	67	70	49	59	35	27	27	31
Three or more years	75	157	206	226	194	167	149	148
Flocks non-reacting (%):								
First year	46.21	57.38	55.06	63.44	62.50	72.97	72.97	72.09
Three or more years	61.98	78.89	81.42	84.01	88.58	93.82	92.55	98.01

the flock-owner because of the expenditure involved. Furthermore, some entertain the opinion that it is practically impossible to eradicate the disease through intensive retesting. Failure to eliminate infection from a flock through short-interval retesting may usually be accounted for by the lack of proper application of sound eradication measures which are an important supplement to short-interval testing in the eradication of the disease.

Table III shows data concerning short-interval testing in ten infected flocks. The flocks represented varied in size from 125 to 2,722 birds. The percentages of infection ranged from 0.54 to 27.34. In one flock no reactors were detected after the first test. In six flocks no reactors were detected after the second test, and in three flocks none after the third test. All flocks tested the subsequent season revealed no reactors in the old birds (tested the previous year) and their progeny (tested for the first time). In Massachusetts short-interval retesting has been successful where it has been conscientiously carried out.

TABLE III—*Retesting data of ten infected flocks.*

FLOCK		FIRST TEST	SECOND TEST	THIRD TEST	FOURTH TEST	FIFTH TEST	RESULTS OF SUBSEQUENT SEASON
1	Birds tested	189	152	48			467
	Reactors (%)	1.59	0.00	0.00			0.00
2	Birds tested	369	256	218			232
	Reactors (%)	0.54	0.39	0.00			0.00
3	Birds tested	125	98	91			201
	Reactors (%)	20.00	4.08	0.00			0.00
4	Birds tested	243	262	223	179		199
	Reactors (%)	11.11	1.15	0.00	0.00		0.00
5	Birds tested	464	444	433	397		1,087
	Reactors (%)	2.37	0.45	0.00	0.00		0.00
6	Birds tested	1,765	1,559	1,508	1,108	767	1,796
	Reactors (%)	3.17	0.13	0.00	0.00	0.00	0.00
7	Birds tested	2,079	1,929	1,811	1,648	1,337	2,132
	Reactors (%)	3.17	1.09	0.00	0.00	0.00	0.00
8	Birds tested	704	691	610	422		693
	Reactors (%)	8.24	8.83	0.16	0.00		0.00
9	Birds tested	2,722	2,413	2,284	1,929		3,707
	Reactors (%)	1.80	0.54	0.48	0.00		0.00
10	Birds tested	640	440	399	352	339	747
	Reactors (%)	27.34	4.32	1.00	0.00	0.00	0.00

Occurrence of Infection in Flocks Previously Non-Reacting

Flocks once free of the disease may become reinfected, as shown in table IV. The origin of reinfection may arise from a variety of sources or a combination of sources which may be regarded as faulty management in disease prevention. However, table IV shows one group of flocks in which the origin of infection was unknown. No definite explanation could be presented as to the source of infection. It is unlikely, although not impossible, that the infection had remained latent in flocks that had been non-reacting for two or more consecutive years. The majority of "breaks" occur in flocks with shortest non-reacting testing history, which illustrates that the owners of such flocks are not fully cognizant of the importance of following effective disease-prevention measures.

Doubtful Reacting Birds

The doubtful reacting bird, when detected in a previously non-reacting flock, is a baffling problem to the diagnostician or control official who interprets the results of the test. No precise definition can be offered for the so-called doubtful pullorum-disease reactor. While some reactions appear definitely atypical or non-specific, others may resemble typical agglutination but occur only in low dilutions. Such reactors may or may not yield *S. pullorum* on bacteriological examination. A great percentage of the atypical and low-titre reacting birds will also react with antigens prepared from other members of the *Salmonella* genus. To what extent other organisms, related or unrelated to *S. pullorum*, may be responsible for these reactions remains a problem for future study. In routine testing the detection of only doubtful reacting birds in a previously non-reacting flock should not be sufficient evidence to condemn the flock as infected. Each year necropsies of doubtful-reacting birds detected in previously negative flocks have revealed that when such necropsies do not show infection it appears safe to regard the flock as negative. The fact is realized that necropsies have their limitations and are not infallible, but to condemn a flock as infected, on the basis of doubtful reactions which may be due to causes aside from *S. pullorum*, does not seem justifiable to the owner who highly values a non-reacting test.

Significance of Other Salmonelloses in Pullorum Disease Eradication

A Salmonella disease which probably ranks second to pullorum infection in chickens is fowl typhoid. However, certain geo-

TABLE IV—*Flock information on "breaks."*

EXPLANATION FOR INFECTION	NUMBER OF "BREAKS"						TOTALS	NUMBER OF CONSECUTIVE YEARS NON-REACTING					
	1931	1932	1933	1934	1935	1936		2	3	4	5	6	7
Introduction of stock from untested flocks	1					1	2	1					1
Introduction of stock from tested infected flocks	2	3	1	2			8	4	4				
Eggs hatched at poultry plants with infected flocks	2						2	1	1				
Birds returned from egg-laying contests	1						1	1					
Combination of sources	2	1				1	4	4					
Unknown	1	3	6	3	3	3	19	5	4	3	4	2	1
Total breaks	9	7	7	5	3	5	36	16	9	3	4	2	2
Total number of negative flocks the previous year	210	210	180	145	156	163							
Percentage of "breaks"	4.29	3.33	3.89	3.45	1.92	3.07							

graphical sections appear to be comparatively free of this disease. Whether the low incidence of fowl typhoid in the New England States can be attributed to natural influences or whether it is due in a large measure to the pullorum-disease eradication which has been operating for many years is difficult to determine at this time. Since chickens infected with *S. gallinarum* will as a rule react when their sera are tested with *S. pullorum* antigen in almost the same degree as with *S. gallinarum* antigen, it is logical to assume that in the routine testing of chickens for pullorum disease some birds will be eliminated which may be affected with either disease. This fact has been observed in the routine testing for pullorum disease in Massachusetts in two different flocks.

In one flock which had passed a negative test for pullorum disease the previous season, one fowl-typhoid reactor was revealed among 688 birds. This bird was a male and exhibited a titre of 1:320 with the routine pullorum antigen. Necropsy revealed a marked pericarditis and epicarditis, and bacteriological examination yielded *S. gallinarum*. The history of this infected bird disclosed that it originally came from a pullorum-clean flock but had been reared on premises with untested stock and was returned to the owner's flock two weeks prior to the test.

The other fowl-typhoid-infected flock consisted of 1,883 birds, which revealed six reactors with pullorum antigen, showing different degrees of reaction. Only from one reactor was *S. gallinarum* isolated. The source of infection was not ascertained. Part of the flock (291 birds) was retested approximately four weeks later and no reactors were detected. The following season this flock, consisting of 1,991 birds, was tested and one reactor (old hen) was detected. Necropsy of this bird yielded *S. gallinarum*. Since this bird had been tested the previous year, it is likely that the infection may have been dormant at the time of the test or acquired since the test. A retest of part of the flock revealed no additional reactors, and a test the following year was 100 per cent negative.

Sera of chickens infected with either *S. pullorum* or *S. gallinarum* may react equally well with pullorum antigen. In geographical sections where these two Salmonella diseases have attained epornitic proportions and routine testing is followed for pullorum disease, the testing agency is confronted with the problem of classifying the positive flocks as to whether they are infected with one or the other of the two diseases or both. From the standpoint of the breeding flock, an accurate determination of these two Salmonella infections matters little, since a flock

infected with either disease should be condemned for breeding purposes, although *S. pullorum* apparently is transmitted more readily through the egg than *S. gallinarum*. In localities where the two Salmonella diseases coexist, it is possible that the difficulty experienced in eliminating the infection which is thought to be pullorum disease, may be due to the fact that it is actually fowl typhoid.

In addition to pullorum disease and fowl typhoid there are other Salmonella diseases which may occur. Chickens infected with other members of the Salmonella group aside from *S. pullorum* and *S. gallinarum* may react with pullorum antigen. This was observed in a flock from which a Salmonella organism was recovered from chicks that exhibited an acute paratyphoid outbreak. When the survivors were approximately seven months of age, they were tested with both pullorum antigen and an antigen prepared from the Salmonella strain originally isolated from the outbreak. With the pullorum antigen one dilution (1:25) was employed, while with the paratyphoid antigen three dilutions (1:25, 1:50, 1:100) were employed. Table V shows the results of the tests.

TABLE V—*Data concerning a flock which was tested with pullorum and paratyphoid antigens.*

DATE OF TEST	BIRDS	REACTORS			
		PULLORUM	BIRDS YIELDING S. PULLORUM	PARATYPHOID	BIRDS YIELDING PARATYPHOID
November 15	1,799	10	0	44	8
December 18	1,705	2	0	44	3
January 15	1,569	2	0	12	1
February 12	1,448	N.T.*	—	7	0
March 12	1,251	N.T.	—	10	0
April 30	818	N.T.	—	13	1

*N.T. = Not tested.

It is observed that a greater number of birds reacted with the paratyphoid antigen than with the pullorum antigen. Four birds, reacting weakly with the pullorum antigen, did not react with paratyphoid antigen on the original test. On the second test only two reacted with the pullorum antigen, both of which also reacted with the paratyphoid antigen. On the third test only one of the two birds reacting with the pullorum antigen reacted with the paratyphoid antigen.

The majority of strong reactors detected with the partyphoid antigen would as a rule also react with pullorum antigen. However, frequent retests of the reactors show that the reactions fluctuate markedly, especially with birds that exhibit a very low titre. The eradication of paratyphoid infection, at least the type encountered in this case which has not been definitely classified, is extremely difficult, if not impossible, by means of the agglutination test. The agglutination test failed to detect three birds from which the organism was recovered. Two of these birds were removed from the flock between the last two tests. However, it is possible that continuous testing and removal of reactors might tend to maintain the degree of infection at a level which would not express itself in the progeny. The progeny of this flock was tested the following fall and while the paratyphoid antigen did detect some reactors, these birds did not reveal Salmonella organisms on necropsy.

Continued observations are being made on this flock and a more detailed report of the Salmonella disease mentioned here will be published. It may be emphasized that paratyphoid infections may be responsible for inaccurate diagnosis of pullorum disease when one resorts only to the macroscopic agglutination test.

Discussion and Summary

The control and eradication of Salmonelloses in chickens is recognized by diagnosticians and disease-control agencies as a tremendous but not an insurmountable, task. The data presented in this paper show that pullorum disease, fowl typhoid, and possibly other Salmonella infections can be eradicated from flocks.

An effective and successful program can be carried out by the use of the macroscopic agglutination test along with sound eradication measures. Flocks should be 100 per cent tested annually. When infection is discovered, a sound retesting program should be followed if the value of the flock warrants the expenditure. Flocks revealing no reactors should be employed as foundation stock for replacing infected flocks. Eggs from flocks revealing no infection should not be incubated on the same premises where eggs from infected or untested birds are incubated. Likewise, pullorum-clean chicks should not be reared with chicks from untested or infected flocks.

Commercial hatcheries which handle thousands of eggs and chicks annually should strive to establish and maintain pullorum-clean flocks. Separate hatching facilities far remote from their main hatching operations should be provided and used only for eggs and chicks which are to replace their custom-breeding flocks.

Such a procedure would in a way be similar to the practices followed in the New England States. This would permit dealing with fewer flocks for replacements, consequently reducing the factor of human error. Only those flocks whose owners are conscientiously disease-eradication-minded should be selected. Such a program supplemented with effective education should expedite pullorum-disease eradication. It is hoped that the widespread interest and participation in pullorum-disease testing will lead to a sound eradication program which is national in scope. Progress in eradication cannot be anticipated from a federal plan which permits the use of ineffective testing procedures and official grade terms that mislead the public. Poultrymen and hatcherymen should appreciate the fact that pullorum disease is a transmissible, infectious disease, whose dissemination can be prevented by practical means which will lead to ultimate eradication of the infection. Federal and state agencies should recognize that competent personnel and proper organization are necessary to execute and carry out a sound eradication program.

REFERENCES

[1]Plastridge, W. N., and Rettger, L. F.: Variants of *Salmonella pullorum*. Jour. Inf. Dis., 1 (1932), pp. 146-161.

[2]Mallmann, W. L.: The dissociation of *Salmonella pullorum* and related species. Mich. Agr. Exp. Sta. Tech. Bul. 122 (1932), pp. 3-40.

[3]Van Roekel, H., and Rettger, L. F.: Variation of *Salmonella pullorum*. Jour. Inf. Dis., lviii (1936), pp. 172-184.

Veterinary Students from Far and Wide

The cosmopolitan character of a veterinary student body is to be expected, since there are only ten veterinary colleges in the United States. At the initiation of eight veterinary students into the honorary society of Alpha Psi, at Michigan State College, on May 14, the diverse origin of the student body was well illustrated. Manuel Arteaga hailed from Cuba; Raymond Bankowski and Edward Schilf came from Illinois; John Arnold from Indiana; James Nundy from New York; Charles Helmboldt from Connecticut; Clifford Westerfield from the blue grass country of Kentucky, and only one—Don Wright—from Michigan.

The first-year veterinary class at Michigan State College includes students from 18 states and the District of Columbia—only two out of nine being residents of Michigan. The pre-veterinary students hail from ten states—about half coming from Michigan. Every section of the country is represented.

W. G.

THE EFFECT OF FEEDING PARADICHLOROBENZENE-TREATED CORN TO SWINE*

By M. W. EMMEL *and* W. W. HENLEY†
Florida Agricultural Experiment Station
Gainesville, Fla.

For many years, paradichlorobenzene has been used as a fumigant for the destruction of corn weevils. The use of this product apparently does not affect the germination of the corn kernel when seed corn is treated. There is evidence to show, however, that paradichlorobenzene-treated corn, when fed to animals, renders the meat unfit for food.

Robbins[1] fed paradichlorobenzene-treated corn to poultry with the result that the meat was distasteful and inedible. Corn so treated and fed to laying hens resulted in a similar condition of the eggs. Marchal[2] observed that feeding paradichlorobenzene-treated grain had no harmful effect upon domestic animals but that the chemical tainted the milk. Dikmans[3] found that the odor of paradichlorobenzene was retained in the flesh of hogs and dogs treated with this agent as an anthelmintic and that its presence was also noted in the eggs of hens fed organs of a dog previously treated with paradichlorobenzene.

The Agronomy Department of the Florida Agricultural Experiment Station had a surplus of paradichlorobenzene-treated seed corn from the 1934 planting season. Since previous experimental data have shown that the meat of swine would no doubt become affected after feeding corn treated with this chemical, it seemed desirable to determine how long the flesh remained in this state and to study any pathology that might develop during the feeding period.

EXPERIMENTAL DATA

Eleven hogs were divided into three groups, August 7, 1934, as follows: One group consisting of five hogs to be fed a ration containing paradichlorobenzene-treated corn for 15 days; a second group of four hogs to be similarly fed for 28 days; and a third group of two hogs which received a ration containing untreated corn. All animals received a full feed or a mixture consisting of 60 parts corn, 30 parts of shorts and 10 parts of fish meal. In instances in which paradichlorobenzene-treated

*Received for publication, October 9, 1936.

†Resigned; now Assistant Animal Husbandryman, U. S. Bureau of Animal Industry, stationed at Gainesville, Fla.

Such a procedure would in a way be similar to the practices followed in the New England States. This would permit dealing with fewer flocks for replacements, consequently reducing the factor of human error. Only those flocks whose owners are conscientiously disease-eradication-minded should be selected. Such a program supplemented with effective education should expedite pullorum-disease eradication. It is hoped that the widespread interest and participation in pullorum-disease testing will lead to a sound eradication program which is national in scope. Progress in eradication cannot be anticipated from a federal plan which permits the use of ineffective testing procedures and official grade terms that mislead the public. Poultrymen and hatcherymen should appreciate the fact that pullorum disease is a transmissible, infectious disease, whose dissemination can be prevented by practical means which will lead to ultimate eradication of the infection. Federal and state agencies should recognize that competent personnel and proper organization are necessary to execute and carry out a sound eradication program.

REFERENCES

[1]Plastridge, W. N., and Rettger, L. F.: Variants of *Salmonella pullorum*. Jour. Inf. Dis., 1 (1932), pp. 146-161.

[2]Mallmann, W. L.: The dissociation of *Salmonella pullorum* and related species. Mich. Agr. Exp. Sta. Tech. Bul. 122 (1932), pp. 3-40.

[3]Van Roekel, H., and Rettger, L. F.: Variation of *Salmonella pullorum*. Jour. Inf. Dis., lviii (1936), pp. 172-184.

Veterinary Students from Far and Wide

The cosmopolitan character of a veterinary student body is to be expected, since there are only ten veterinary colleges in the United States. At the initiation of eight veterinary students into the honorary society of Alpha Psi, at Michigan State College, on May 14, the diverse origin of the student body was well illustrated. Manuel Arteaga hailed from Cuba; Raymond Bankowski and Edward Schilf came from Illinois; John Arnold from Indiana; James Nundy from New York; Charles Helmboldt from Connecticut; Clifford Westerfield from the blue grass country of Kentucky, and only one—Don Wright—from Michigan.

The first-year veterinary class at Michigan State College includes students from 18 states and the District of Columbia—only two out of nine being residents of Michigan. The pre-veterinary students hail from ten states—about half coming from Michigan. Every section of the country is represented.

W. G.

THE EFFECT OF FEEDING PARADICHLOROBENZENE-TREATED CORN TO SWINE*

By M. W. EMMEL *and* W. W. HENLEY†
Florida Agricultural Experiment Station
Gainesville, Fla.

For many years, paradichlorobenzene has been used as a fumigant for the destruction of corn weevils. The use of this product apparently does not affect the germination of the corn kernel when seed corn is treated. There is evidence to show, however, that paradichlorobenzene-treated corn, when fed to animals, renders the meat unfit for food.

Robbins[1] fed paradichlorobenzene-treated corn to poultry with the result that the meat was distasteful and inedible. Corn so treated and fed to laying hens resulted in a similar condition of the eggs. Marchal[2] observed that feeding paradichlorobenzene-treated grain had no harmful effect upon domestic animals but that the chemical tainted the milk. Dikmans[3] found that the odor of paradichlorobenzene was retained in the flesh of hogs and dogs treated with this agent as an anthelmintic and that its presence was also noted in the eggs of hens fed organs of a dog previously treated with paradichlorobenzene.

The Agronomy Department of the Florida Agricultural Experiment Station had a surplus of paradichlorobenzene-treated seed corn from the 1934 planting season. Since previous experimental data have shown that the meat of swine would no doubt become affected after feeding corn treated with this chemical, it seemed desirable to determine how long the flesh remained in this state and to study any pathology that might develop during the feeding period.

EXPERIMENTAL DATA

Eleven hogs were divided into three groups, August 7, 1934, as follows: One group consisting of five hogs to be fed a ration containing paradichlorobenzene-treated corn for 15 days; a second group of four hogs to be similarly fed for 28 days; and a third group of two hogs which received a ration containing untreated corn. All animals received a full feed or a mixture consisting of 60 parts corn, 30 parts of shorts and 10 parts of fish meal. In instances in which paradichlorobenzene-treated

*Received for publication, October 9, 1936.

†Resigned; now Assistant Animal Husbandryman, U. S. Bureau of Animal Industry, stationed at Gainesville, Fla.

corn was fed, the corn in above mixture was so treated. The animals receiving treated corn were weighed at the start and conclusion of the 15- and 28-day periods. These animals were then placed on a ration containing untreated corn and similar weights recorded. The animals in each lot were killed at certain intervals to determine the edibility of the meat.

Results: The experimental data are recorded in table I. Two animals, one in each of the two groups fed treated corn, were killed at end of 15- and 28-day feeding periods. The meat was distasteful and inedible. At this time, the remaining animals in these two groups were placed on untreated corn. In those animals that received treated corn for 15 days, it required 62 days for the meat to become edible, while in those fed treated corn for 28 days, 85 days were required. The average daily gains of animals fed treated corn for 15 days ranged from 0.466 to 0.833 pounds; one animal lost 1 pound during the 15 days. The animals receiving treated corn for 28 days made an average daily gain of from 0.035 to 0.732 pounds. After being placed on untreated corn, both groups made more satisfactory gains, the average daily gain ranging from 1.044 to 1.508 pounds. The two control animals made an average daily gain of 0.741 and 1.473 pounds, respectively.

Macroscopic lesions: The myocardium was soft and flaccid. The liver and kidney showed cloudy swelling. All other organs appeared normal.

Microscopic lesions: All of the smooth muscle of the spleen showed cloudy swelling, which was usually more pronounced in the trabeculae than in the capsule. Cloudy swelling was apparent also in the smooth muscle of the larger arteries. There was slight general congestion, more marked near the capsule of the organ.

General cloudy swelling of the hepatic cells was observed in all cases. In the animal to which paradichlorobenzene-treated corn was fed four weeks and which was killed at the end of that period, cloudy swelling was more marked near the periphery of the lobules, many of the hepatic cells being more or less devoid of cytoplasm.

In the kidney, the epithelium of groups of tubules and occassionally single tubules showed cloudy swelling. The lumen of the tubules usually contained much débris. There were a few scattered, small foci of proliferation of histiocytes in the intertubular tissue. Small areas of congestion often occurred, particularly in the medullary portion of the organ. Occasional glomeruli

TABLE I—*Experimental data on pigs paradichlorobenzene-treated corn.*

PIG	DAYS FED TREATED CORN	INITIAL WEIGHT*	GAINS WHEN FED TREATED CORN			DAYS FED UNTREATED CORN BEFORE SLAUGHTER	GAINS WHEN FED UNTREATED CORN			ODOR OF PARA-DICHLORO-BENZENE	EDIBLE OR INEDIBLE
			FINAL WEIGHT	GAIN	AVERAGE DAILY GAIN		FINAL WEIGHT	GAIN	AVERAGE DAILY GAIN		
4	15	92.5	100.5	8.0	.533	0	—	—	—	Strong	Inedible
3		88.0	95.0	7.0	.466	13	112.0	17.0	1.307	Strong	Inedible
5		75.5	74.5	—1.0	—	34	110.0	35.5	1.044	Moderate	Inedible
1		98.0	110.5	12.5	.833	48	166.0	55.5	1.156	Faint	Inedible
2		88.5	96.5	8.0	.533	62	190.0	93.5	1.508	None	Edible
10	28	63.0	64.0	1.0	.035	0	—	—	—	Strong	Inedible
6		86.0	106.0	20.0	.714	21	132.0	26.0	1.238	Strong	Inedible
8		101.5	122.0	20.5	.732	49	194.0	72.0	1.469	Strong	Inedible
7		62.5	68.0	5.5	.196	85	180.0	112.0	1.317	None	Edible
9	None	102.5	—	—	—	56	185.0	82.5	1.473	None	Edible
11		58.5	—	—	—	56	102.0	43.5	.741	None	Edible

*All weights expressed in pounds.

were enlarged; most of the glomeruli showed evidence of congestion.

Congestion was observed in scattered areas in the lungs. Cloudy swelling was present in the smooth muscle of both the bronchi and larger arteries.

The myocardium was in a state of cloudy swelling and slight edema often occurred between the muscle bundles.

Cloudy swelling was observed in the circular and longitudinal muscle coats of the intestines. The longitudinal muscle coat in particular was irregularly thickened; slight edema occurred in scattered areas. The surface as well as the glandular epithelium showed cloudy swelling. In many areas, particularly at the tips of the villi, necrosis occurred. The stroma of the villi often showed edema and small foci of proliferation of histiocytes.

Necrosis of the surface epithelium of the stomach was observed in many areas; the adjacent tissue usually was slightly edematous. The remaining surface and glandular epithelium was in a state of cloudy swelling. Much of the glandular epithelium, particularly in cases where microscopic lesions in other organs were pronounced, was almost devoid of cytoplasm. Small areas of congestion were observed in scattered areas near the surface of the mucosa. Cloudy swelling was present in the muscle coats of the stomach.

The mesenteric lymph-nodes showed scattered areas of congestion.

Cloudy swelling was observed in the parenchymatous tissue of the salivary, thymus and thyroid glands as well as in the pancreas.

DISCUSSION

The experimental data indicate that the flesh of pigs fed paradichlorobenzene-treated corn becomes tainted with paradichlorobenzene in less than 15 days. A period of 62 days was required for pork to become edible after untreated corn was fed to hogs that had received treated corn for a period of 15 days. In animals fed treated corn for 28 days, a period of 85 days elapsed during which untreated corn was fed before the pork became edible.

Animals fed paradichlorobenzene-treated corn failed to make gains equal to those made by the two control animals which received untreated corn. After the two groups of experimentally fed animals were placed on untreated corn, they made more satisfactory gains.

The macroscopic lesions observed indicate that paradichlorobenzene-treated corn produces pathology which might easily be overlooked. The microscopic lesions, however, were present in all cases and seem to vary in intensity according to the length of time treated corn was fed. When the feeding of treated corn was discontinued and the animals were fed untreated corn, the lesions decreased in intensity but were still present in all cases at the conclusion of the experiment.

The presence of the microscopic lesions observed probably explains why the animals fed treated corn failed to make gains comparable to the gains made by the control animals. These results do not agree with those of Marchal, from which he concluded that the feeding of paradichlorobenzene-treated grain had no harmful effects upon domestic animals.

SUMMARY

The meat of animals fed 15 days on paradichlorobenzene-treated corn was distasteful and unfit for food. The length of time required for the meat to become edible after a 15-day period on treated corn was approximately 62 days; after a 28-day period on treated corn, this period was approximately 85 days. Cloudy swelling of the heart, kidney and liver was the only macroscopic lesion observed. Microscopic lesions consisted chiefly of cloudy swelling in all of the parenchymatous organs and involuntary muscle.

REFERENCES

[1]Robbins, F. E.: Effect of feeding paradichlorobenzene-treated corn to poultry. Jour. Econ. Entom., xv (1921), p. 509.
[2]Marchal, M.: The effect of grain treated with paradichlorobenzene or chloryl upon domestic animals. Chem. Absts., xx (1926), p. 2550.
[3]Dikmans, G.: Jour. Agr. Res., xxxv (1927), p. 7.

Veterinarian Interested in Palominos

According to a recent report in *The Cattleman*, Dr. Verne A. Scott, veterinarian at the John Tarleton Agricultural College, Stephenville, Texas, has purchased five Palominos. These horses are of golden color with ivory-white mane and tail. They are descendants of mares and stallions sent to Mexico when it was under Spanish rule of Queen Isabella. The offspring of these imported horses became famous and were so highly prized that it was considered sacrilege to sell them. Only as a gift from one cabellero to another, could one of these animals change ownership, and even today, no fiesta in Mexico or California is complete without a Palomino.

LAMENESS IN THE HORSE, WITH SPECIAL REFERENCE TO ACUTE LAMINITIS*

By N. D. BACKUS, *Elyria, Ohio*

I feel that all veterinarians are confronted with the same problems in everyday general practice that I am, and know just as much about diagnosis and treatment.

We have what I term "seasonable" diseases so, at certain seasons of the year, one can often sit at his telephone and make a probable diagnosis before he leaves his office. In fact, I know of some veterinarians who carry with them only limited supplies of drugs. After receiving a call, the veterinarian prepares a bottle or two of medicine and leaves the office thoroughly equipped to treat whatever he may find upon his arrival, even before the examination of his patient. It has been my experience that the "arm-chair" diagnosis is often in error. I try to equip myself so that I may be able to cope with whatever condition I find after careful examination of the patient.

Correct diagnosis is the key to the successful career of a practitioner. It requires in many cases all the learning and common sense which he has to apply. As our patients are unable to talk to us, we must be very capable of following the history, which is not always reliable, as important points often are omitted by the owner or caretaker, sometimes intentionally and sometimes otherwise. It is intentional, in some cases, when the animal has been treated by the owner prior to calling the veterinarian, or by some helpful neighbor or a member of the Termite Tribe called "quack." Many times the veterinarian is not careful enough in asking the owner many questions which will bring out helpful history and aid in making a final conclusive diagnosis of the case. When you are thoroughly satisfied with your diagnosis, the treatment to be administered is not difficult. Almost every catalog received from the pharmaceutical and biological firms contains a list of preparations intended for use in various conditions. The most satisfactory method for me is to follow the use of drugs which I was taught to use in my study of medicine at college.

I have not said anything so far about the horse, which was the subject assigned to me, and there are so many things one could discuss that it leaves only a narrow path to follow. As I said in the beginning, we have what are termed "seasonable" diseases. I mean by that, in the beginning of the year, the live stock ship-

*Presented at the semi-annual meeting of the Michigan-Ohio Veterinary Medical Association, Blissfield, Mich., October 29, 1936.

per begins to bring in a few carloads of horses, so we then have many conditions, such as influenza, strangles and pneumonia. In the spring, we get azoturia, lymphangitis and some digestive troubles. Later in the season, after haying and harvesting, we have a run of digestive troubles when the animals are fed new hay and new oats. These digestive troubles are more or less prevalent, so at this season of the year, there are many calls from owners stating that their horses are suffering from colic. However, it is not known what may be found upon arrival, as colic covers all kinds of abdominal pain.

On this subject of abdominal disturbances in the horse, I am often at a loss to make a satisfactory diagnosis, at least to myself. The horse shows evidence of pain by pawing, getting up and down, rapid breathing, accelerated pulse and looking back at his side, peristalsis, weak or absent on one or both sides, a metallic tinkling in the bowels, feces may be soft, formed or absent, and all the other symptoms which you have seen many times. What has always interested me most is to be able to make a differential diagnosis. The same set of symptoms may mean one of many conditions which may cause distended stomach, colon or cecum, obstruction of the small bowel or the ileocecal region, impaction, volvulus, hernia, intussusception, rupture, tumor, abscesses, ruptured diaphragm, and many other things which one will discover upon postmortem examination. I trust that some one will enlighten me on how to differentiate these conditions.

We have one condition which is not seasonable, but continuous. It is lameness in the horse and I have been unable to find many books on this subject, although it is one of the most frequent ailments we have to treat. If you will bear with me for a few moments, I will endeavor to tell you some of the things which experience has taught me in diagnosis, differential diagnosis and treatment of one condition which I am going to discuss more fully, namely, laminitis.

While the older men in the profession have, no doubt, had a much wider experience, the younger men may gain a few helpful hints. Some of the most perplexing conditions with which I have met are with the race or running horses. I have had considerable experience with harness horses. To begin with, I believe a definition of soundness is in order. The best definition of a sound horse I have ever heard came from our good friend, Dr. Harry Fulstow, of Norwalk, Ohio. It is as follows: "A horse which is free from all disease, has nothing about him which will interfere or is likely to interfere with his usefulness or that will depreciate his value."

All that is known on the subject of lameness is based on a knowledge of anatomy and the part it plays in locomotion. Without this knowledge, no one can master the principles necessary in the diagnosis of lameness. It has always been my policy to make a thorough visual examination of the animal before I approach him for the physical examination. While making the visual examination, one can endeavor to secure the history which is of importance. Several questions may be asked to assist in the diagnosis, namely:

1. How long has the animal been lame?
2. Under what conditions did he go lame?
3. Was the lameness first noticed when at work or at rest?
4. Has he been shod recently?
5. Did you ever notice any swelling?
6. Has the lameness increased since you first noticed it?
7. Does the lameness increase upon exercise?
8. What has been done in the way of treatment?

In cases of slight lameness, careful study, practice and experience are imperative. Severe lameness is readily recognized. Even at rest, the animal may show distinct symptoms of pain, such as pointing and raising the affected foot or leg. By pointing, I mean the raising of the foot, either in front or behind, to the inside or outside of an imaginary vertical line drawn from the shoulder to the ground. The animal's instinct leads him to place his foot in a position that will relieve the pain. If lameness is not severe, the animal is either walked, trotted, ridden or driven. If no conspicuous irregularity in the gait is seen, the ear may be of value in making a diagnosis. The louder hoof-beat will come from the sound leg; the lame leg hits the pavement or ground more lightly, producing an unequal sound. Some horses, beyond a certain speed, hop behind. This hop disappears when the speed is reduced or the animal is being led.

If nothing outstanding is in evidence, I always start at the foot. When making an examination, one must consider the temperament of the individual. Some animals are supersensitive, so one must be careful not to confuse this condition with actual sensitiveness when palpating or tapping on the foot. Frequently, you can tap the opposite foot to determine this condition. By comparison of different parts of the same member or with an analogous portion of another member, any abnormal condition can be detected. Personally, I have never been able to detect sufficient difference in the temperature of one part from another to be of any assistance to me.

Throbbing of the digital arteries is often of assistance in detecting laminitis. The sense of touch must be trained to examine a tendon for the presence of a swelling, or the bones for ringbone, splints and other conditions. This sense will assist greatly in arriving at a definite and proper conclusion when digital manipulations have been employed. The necessary qualification which the diagnostician must possess is that of being able to judge carefully the nearness of any given exostosis to articular structures. Also the extent or area of the base as well as its exact position needs to be determined before one may estimate the probable outcome in any case where treatment may be encouraged or discouraged by the practitioner.

Ringbone may, because of the size or location of the exostosis, constitute a condition which cannot be relieved in any way. Because of the distribution of the osseous deposits, it may respond to treatment with satisfactory recovery. In many cases, when the diagnosis is difficult, diagnosis by elimination is resorted to and, as in any other case where the recognition of the cause is difficult, exclusion of the existence of conditions, one at a time, by analysis of symptoms, often enables the practitioner to eliminate all but the disturbing element.

The x-ray has added to our equipment a most valuable and satisfactory aid in diagnosing fractures and other conditions of the bony structures where exostoses or adhesions cannot otherwise be determined.

So much for a brief résumé of diagnostic methods. As time will permit me to take up only one condition, I am going to discuss acute laminitis, a condition which is often uncertain as far as a prognosis is concerned and in my opinion may be confused, especially in the light harness horse, with a condition which I term myositis. In this condition, the animal may show almost the identical symptoms with varying severity.

Laminitis at the onset may be confused by the beginner as being a case of pleurisy. Laminitis is primarily a non-infectious inflammation of the sensitive laminae, which frequently affects the front feet. Sometimes all four feet are affected and, less frequently, one foot, and rarely the hind feet alone. There is a spontaneous and diffuse inflammation of the whole of the sensitive structures of the foot, more particularly the sensitive laminae.

Causes of Laminitis

Strictly speaking, traumatic laminitis, as referred to by some authors, gives rise to a limited inflammation of the sensitive

laminae, so it is a limited laminitis and may be caused by a nail-bound shoe, the nails having been driven too closely to the sensitive laminae, by corns, or other abnormalities. This is not laminitis proper but compares closely to it. One may theorize on the cause and enumerate many conditions which are apparently contributory towards producing the affection. It is stated by some authors and may, in the present state of our knowledge, be vaguely described as a poisonous condition of the blood-stream. This, as clinical evidence teaches us, may result from a variety of causes. These causes may be grouped so a more definite understanding is possible as a result of such a grouping. We would include conditions which directly or indirectly affect the digestive tract, such as drinking a large quantity of cold water, being caught in a rain when the body is warm, and excessive feeding with too stimulating or too irritating a diet. Certain foods seem more prone than all others to bring on an attack of laminitis—wheat, malt, corn, rye, peas, beans and sometimes new oats. In many cases, we find digestive disorders are accompanied by increased peristalsis and often a diarrhea. These causes are construed, by some authors, to be due to the effect of the toxic material entering the blood-stream and affecting the vaso-constrictor nerves. In parturient laminitis, the same reason is given. There is sufficient toxic material taken up to affect the vaso-constrictor nerves and cause a localization in the sensitive laminae of the feet.

In addition to the above-mentioned feeds, many other causes may be enumerated, especially in some feeding materials which are mixed and placed on the market. Another cause of laminitis is a severe and continued inflammatory condition of the system, such as prolonged attacks of pneumonia, shipping fever, and metritis following parturition. I believe parturient laminitis offers more conclusive evidence of the absorption of toxic material than any other condition. You often find a small portion of retained placenta or an infected condition of the uterus as evidence. Prolonged or excessive work upon a hard road is apt to induce an attack, or in cases of injury to another leg or foot, the animal persistently standing on the sound foot may cause laminitis. I have seen laminitis in horses, after being turned out to pasture, when the feed was heavy so that the animals would engorge themselves to excess, especially on clover or alfalfa. Influence of the season is to be considered, as it is most frequent in hot, dry weather, especially if the horse's feet are not well cared for and are hard and dry. The general supposition is that the exciting cause is a toxic condition of the blood-stream. The con-

dition does not account for direct injuries which cause a non-infectious inflammation of the sensitive laminae. Dietetic causes are, by far, the most common. In making inquiries as to the history, the veterinarian should always make careful inquiry regarding diet.

Symptoms and Differential Diagnosis

Laminitis is always ushered in with a more or less well-defined set of symptoms: high fever; temperature 106-8° F.; pulse 80-90; respiration, accelerated and short; muscular tremors; mucous membranes injected, especially of the eye, and mouth, hot and dry. Bowels are frequently torpid and the urine is scanty. Yet, the gait may be fairly sound. When first called, in the early stages, it may be rather perplexing and hard to differentiate from enteritis, lymphangitis, pneumonia, pleurisy, peritonitis and myositis. Usually upon being turned abruptly from side to side, the horse will show evidence of pain, so will pick up the feet with a jerky movement which indicates pain. If seen a few hours later, the sensitive laminae have become injected with the inflammatory exudate which causes a compression of the sensitive structures within the hoof, so no mistake need to be made. If the horse is forced to move, the evidence of pain partially subsides, but after the patient is allowed to rest, the pain is more severe than before. If the pain is confined to the two front feet, the hind feet are placed well under the central portion of the body to support the weight; the front feet are advanced in front of a perpendicular line so as to lessen the weight they must bear. If forced to move, the horse assumes the same position, so in turning it uses the hind feet as a pivot, bringing the front feet around by placing as little weight as possible on them. The back is arched, so sometimes the condition is misinterpreted as a disease of the lumbar region. When all four feet are involved, it is very difficult for the animal to move. Frequently the pain is so severe that the horse will show spasms which are sometimes confused with the spasms of tetanus. If the animal is down, it is almost impossible to force it to rise.

When laminitis affects the hind feet, the front feet are placed back towards the center of the body and the hind feet well forward, bringing all four feet closely together. The position of the feet is of great importance, so should not be overlooked. The course of the disease and its pathological anatomy are classified by Reeks under three headings:

1. The period of congestion.
2. The period of exudation.
3. The period of suppuration.

Period of congestion: At the onset, we get the period of congestion, which is an engorgement of the blood-vessels of the laminal portion of the foot. It is engorged with dark venous blood, so hemorrhages of the laminal vessels occur, allowing the infiltration of the surrounding connective tissues which may completely destroy the union between the sensitive and horny laminae. Most commonly, the suppuration is at the toe, which causes what is termed "seedy toe." The toe turns up and the animal walks on his heel.

Period of exudation: The exudation follows congestion and engorgement. This, even more than the congestion, tends to destroy the continuity between the sensitive and the horny laminae, which often leads to almost complete separation when the whole of the body weight is placed on the sole, which is not intended to bear the strain, so it naturally sinks. With the sinking is a corresponding dropping of the os pedis. We get a dropped sole and a marked depression at the coronet. This may be noticed at about the third or fourth week after a severe attack. We get a backward displacement of the os pedis, which may be accounted for in two ways:

(a) The greater vascularity of the membranes covering the anterior surface of the os pedis leads to a greater outpouring of exudate in that portion of the foot.

(b) The manner in which the animal places his weight on the foot, namely on the heel, has a tendency to relax the extensor pedis tendon so that the tension on the flexor perforans is greatly increased. This is the condition which exists when we get a dropped sole.

Period of suppuration: Suppuration sometimes follows the two previous-mentioned stages. We get suppuration, which may break at the coronet or an opening made in the sole. The condition, in my experience, usually is very serious and the results of treatment unsatisfactory.

Chronic laminitis is usually a sequel to an incomplete recovery of the acute form.

One is almost justified in stating that the diagnosis of laminitis offers little or no difficulty. However, in the early stages, it may be confused with another condition which I have encountered in the harness horse, namely, myositis. The animal exhibits very similar symptoms with a history much the same, but these may be differentiated by the lack of extreme sensitiveness upon tapping the feet and the absence of pulsation of the digital arteries. I have found this condition in race horses that had been improp-

erly cared for after a race. It sometimes follows jogging on hard or uneven ground or pavement; excessive work at the beginning of the training season and on long trips in a truck, especially if the animal is of a nervous temperament. The animal walks stiff; does not pick his feet up from the ground; stumbles along; arches his back and shows some discomfort in turning. The temperature is slightly elevated; there is loss of appetite, and the animal will not extend himself when driven. This condition usually responds to treatment in about three to five days, with very few exceptions.

Treatment

Treatment depends, in its early stages, upon the cause. In all cases where digestive disturbances exist, unloading of the alimentary canal is indicated. When the history indicates an overloaded stomach and bowels from the various foods mentioned and no superpurgation has taken place, I administer ¼ to ½ grain of arecoline, hypodermically, in two doses 12 to 18 hours apart; give one to two quarts of mineral oil through a stomach tube, to be followed with one-ounce doses, every two or three hours, of a preparation which I make by using 1 pound of hexamethylenamine, 10 ounces of potassium acetate, 3 ounces of fluid extract of aconite, 8 ounces of fluid extract of bryonia and enough water to make one gallon. This makes a mixture in which each ounce contains 1 dram of hexamethylenamine, ½ dram of potassium acetate, 12 minims of aconite and 30 minims of bryonia. I alternate this mixture with a potassium nitrate and alum solution—each ounce containing 10 grains of potassium nitrate and 60 grains of alum.

When a diarrhea is in evidence, I omit the arecoline and sometimes the oil, but give instead 2 to 4 ounces of sodium hyposulfite, which is a good intestinal antiseptic. I also administer from 50 to 100 cc of normal horse serum every 12 to 24 hours for two or three doses.

Keep the animal in a well-bedded box-stall, put cold packs on the feet and, if possible, remove the shoes before placing the feet in packs. In administering treatment, our first consideration is to remove the cause. Stop the absorption of the toxin in the intestinal tract which is causing or has caused the condition. Cold water is one of the most essential requirements to reduce the congested condition and promote recovery. When a stream of water is available, tie the horse in the stream for three or four hours at a time, if the animal can stand that long. Then rest him for an hour or so and repeat. The water cannot be too cold.

In cases of myositis, the same medicinal treatment, except the arecoline, which may be omitted, will usually have the animal back at work within three to five days.

Court Upholds Government Stand on Gauze Bandages

The stand of the federal government that gauze bandages, represented as "sterile" or "sterilized," should in fact be free from germs when purchased by the ultimate consumer, has been upheld in court. The Federal Court for the Southern District of New York (New York City) has decided that the bandages of a prominent manufacturer are drugs, subject to regulation under the federal Food and Drugs Act, and that because they were contaminated with bacteria they were both adulterated and misbranded.

When the bandages in question were seized by federal authorities, counsel for the claimant contended that such articles were not "drugs" within the definition of that term, as used in the federal Food and Drugs Act, and that therefore the law does not apply to bandages. The law defines as a drug "any substance or mixture of substances intended to be used for the cure, mitigation or prevention of disease."

The government presented evidence to show that gauze bandages are used for packing and dressing wounds, draining infected wounds, packing to arrest hemorrhage, and various other similar purposes. The Court held that gauze bandages rightly come within the statutory definition of "drugs." Especially is this true, held the Court, of a bandage labeled, "Prepared for Surgical Use," as in this case.

The decision, which judicially defines a bandage as a drug, gives support to the campaign carried on during the past year against unsterile articles of this sort being marketed as sterilized. During that period, there have been seized, on instructions of the Food and Drug Administration of the U. S. Department of Agriculture, 5,500 packages of absorbent cotton, 6,800 packages of bandages, 28,000 adhesive compresses, 90 packages of adhesive plaster, 35 first-aid kits containing unsterile cotton and bandages, 6,000 gauze pads, and four boxes of unsterile surgical sutures, according to a recent announcement.

It is not allowable for any one to be judge in his own cause.—Coke.

OVINE ENCEPHALOMYELITIS ASSOCIATED WITH LISTERELLA INFECTION*

By ERWIN JUNGHERR, *Storrs, Conn.*
Department of Animal Diseases
Storrs Agricultural Experiment Station

Although the number of sheep in Connecticut has suffered a considerable decrease during the period 1930 to 1935, according to the latest U. S. census of agriculture, the College still maintains an excellent flock of about 200 pure-bred sheep for teaching and demonstration purposes. The general health of the flock has been good, and the sporadic losses that have occurred could be attributed to parasitism and metabolic disturbances. More recently, however, several of the best animals succumbed to a malady which was accompanied by cerebral symptoms and defied immediate diagnosis. Following the discovery of distinctive histologic lesions in the brain of affected animals, an organism of the genus *Listerella* was isolated which was capable of producing a pathologic picture in experimentally injected animals that resembled the original.

LITERATURE

Owing to their pathogenic range and affinity for the central nervous system, organisms of the genus *Listerella* command considerable interest; they have not been studied extensively. The type species was discovered by Murray, Webb and Swann,[1] in England, as the etiologic agent of an epizoötic disease of laboratory rabbits and guinea pigs which was characterized by emaciation without diarrhea, abundant exudate in the serous cavities and focal necrosis of the liver. Brain involvement was not recorded. On account of the mononuclear leukocytosis observed, the name *Bacterium monocytogenes* was proposed. In the course of plague studies in South Africa, Pirie[2] isolated a similar organism, known as the "Tiger River bacillus," from gerbilles (*Tabera lobengulae*), and found it to be pathogenic for small, but not for large, veld rodents. Unable to classify the organism definitely, he suggested that the words "do not attack carbohydrates" and "facultative" be omitted from the then current definition of the tribe *Kurthiae*, and that it be included in this tribe under the name *Listerella hepatolytica* gen. and sp. nov. Due to the priority of Murray's specific name, the organism is now recognized as *Listerella monocytogenes* in Bergey's Manual.[3] A similar organ-

*Received for publication, November 18, 1936.

ism has since been isolated by Nyfeldt[4] from a case of infectious mononucleosis (angina monocytotica) in man. This organism, when studied by Anton[5] in rabbits, was found to produce a mononuclear reaction resembling that of infectious mononucleosis in man (lymphaemoides Druesenfieber) on intravenous injection, and a conjunctivitis on ocular application. Its suggested etiologic relationship to infectious mononucleosis in man and to spontaneous conjunctival folliculosis in rabbits[6] has not been substantiated, however.

What appears to be the first recorded evidence of disturbance of the central nervous system by *Listerella* organisms was reported by Gill,[7, 8] who found the "New-Zealand ovine encephalitis or X organism" regularly associated with cases of "circling disease" in sheep. The nature of the lesions in field cases suggested to this author that the organism occurred in combination with, or secondary to, a neurotropic virus. Proof for this assumption could not be obtained. *Listerella* organisms were found to be associated with sporadic cases of bovine encephalitis by Jones and Little.[9] Schultz *et al*[10] isolated a similar organism from the spinal fluid of a non-fatal human case of meningoencephalitis for which the name *Corynebacterium parvulum* was suggested. Seastone[11] reported the isolation of *Listerella* organisms from cases of myocardial necrosis in chickens; he made a comparative study of human, bovine, ovine (isolated by Dr. Ten Broeck) and avian strains and found them to be indistinguishable by cultural and serologic means, with the exception of a type strain of *B. monocytogenes* Murray which showed antigenic differences. The monocytic reaction could be produced with all zoölogic varieties, and hence was not peculiar to the rabbit organism. More recently, Burn[12, 13] reported a series of encephalitis cases in man of which three occurred in new-born infants and one in an adult. He[14] found his four human strains to be serologically identical and regarded them as constituting a new species.

Observations

The particular lot of sheep in which the cases occurred were housed in five adjoining pens connected by an alley-way.

Case 1: In January, 1934, a yearling Shropshire ram from pen 4 exhibited droopiness and unilateral conjunctivitis. The animal was subjected to examination following venesection. Autopsy showed the body to be in a good state of nourishment. The right eye manifested a slight muco-purulent conjunctivitis and keratitis which appeared to be of traumatic origin. The heart muscle was flabby; the liver revealed mild cysticercosis, and the aboma-

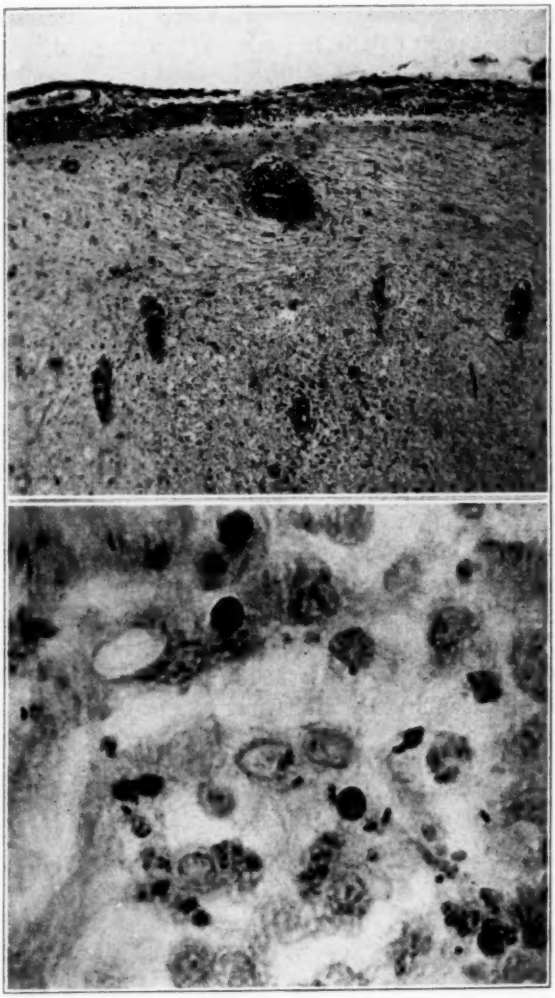


FIG. 1 (above). Medulla of field case showing monocytic infiltration in meningi and perivascular areas (x 70).

FIG. 2 (below). Gram-stained section of figure 1, showing Listerella-like rods (x 900).

304587

sum hemonchosis. The rumen was filled with an unusually large amount of dehydrated ingesta, and the pia-arachnoid of the brain showed fairly extensive pigmentation, but no other gross lesions. Qualitative chemical tests indicated severe albuminuria. Subsequent histologic study of the brain revealed marked perivascular monocytic reaction in the medulla oblongata and similar lesions in the regional meningi (fig. 1); a year later, when the etiologic significance of *Listerella* became apparent, Gram-positive organisms of the general morphology of *Listerella* could be demonstrated in sections of the medulla (fig. 2).

Case 2: On December 20, 1935, a three-year-old Shropshire ewe from pen 5 died, after having shown symptoms of lameness in one hind leg, anorexia, fever, and retraction of the head for not more than 24 hours. The carcass was not submitted for necropsy. In view of the characteristic syndrome and of subsequent developments in the flock, this case should be considered a part of the epizoötiologic picture of the outbreak.

Case 3: A week later, a 10-month-old Shropshire female lamb from pen 5 exhibited first-degree lameness in the left posterior extremity and an unusual tendency to turn its head to the left. Six hours later, the lamb was prostrate in a sternal position, with the head laterally deflected. Postmortem examination of the sacrificed animal revealed a normal state of nourishment, but esophagostomiasis of the colon and minute hemorrhages along the caudal margin of the spleen. Recalling the case of encephalomyelitis of about one year ago, the writer made histologic examination of the brain, with the result that numerous centrally located foci of polynuclear leukocytes were demonstrated in the medulla (figs. 3 and 4). **Aside from the granulocytic** lesions, a marked monocytic reaction was observed in the Virchow-Robin spaces of the vessels, especially in the peripheral area of the medulla, and to a certain extent in the pia arachnoid of the caudal corpora quadrigemina. Sections stained according to Gram showed groups of Listerella-like organisms in the polynuclear foci of the medulla. Numerous other blocks taken from the brain failed to reveal abnormalities by ordinary histologic methods. Routine cultural examination of the internal organs likewise resulted in negative findings.

Case 4: On January 9, 1936, a two-year-old Dorset ewe in pen 1 exhibited frequent turning of the head and general restlessness. At that time the appetite seemed unimpaired. Seventeen hours after the first symptoms were noted, the ewe staggered, the head was held in a horizontally extended position, and a protruding tongue caused the animal to drool. The pupillary reflexes

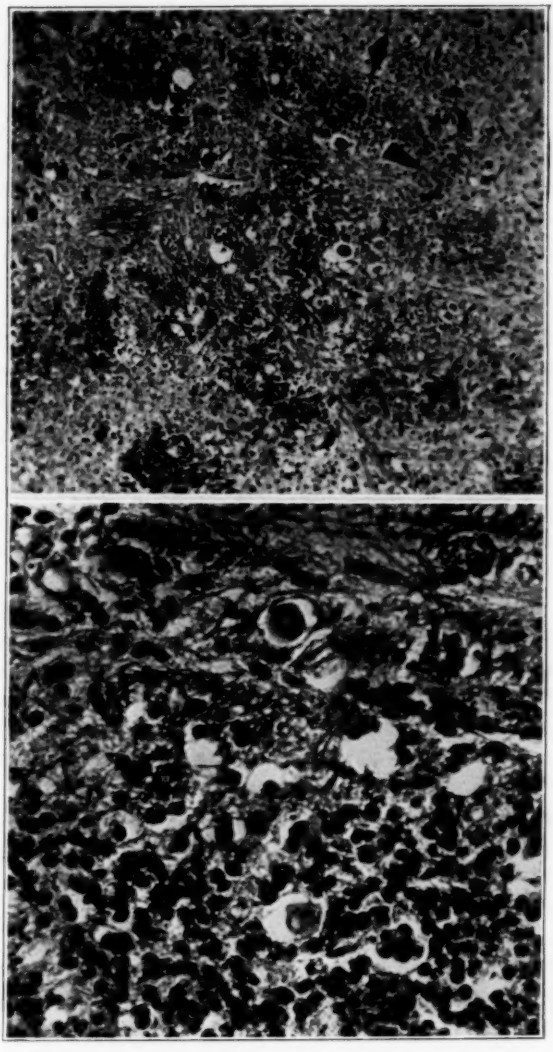


FIG. 3 (above). Medulla of field case showing polynuclear foci. Note necrosis of large ganglion cells in foci (x 70).

FIG. 4 (below). Edge of a focus from figure 3. Note polynuclear and monocytic elements in focus (x 400).

were diminished; the rectal temperature was 105° F., and the differential leukocyte count of the blood was within normal limits. When the animal appeared to be comatose, it was sacrificed for examination. The ventral cervical region exhibited a slight gelatino-hemorrhagic lesion in the subcutis, probably of traumatic origin. The abdominal cavity contained a small amount of transudate and the omasum and abomasum voluminous doughy ingesta. Fecal examination suggested moderate infestation with *Hemonchus contortus, Trichuris ovis, Moniezia sp.*, and coccidia. No evidence of albuminuria was obtained. Histologic examination of the internal organs was essentially negative, although the liver cells showed slight parenchymatous and hydropic degeneration and fatty metamorphosis. Medullary lesions of the granulocytic and monocytic type described in case 3 were marked and definite.

Since the granulocytic foci in the medulla of the previous case suggested a microbic origin of, or an association with, the disease, cultural studies of internal and cranial organs were conducted on a variety of media, including blood-agar slants and boiled egg-meat medium. Save for a few pigmented coccus colonies, the only significant growth was obtained from the medulla on the two media mentioned. This was identified later as a member of the genus *Listerella.*

Case 5: On January 11, 1936, a three-year-old Shropshire ewe in pen 2, known to be in an advance state of pregnancy, was found lying prone, with the head deflected to the left. When excited, the animal reacted by pawing, grinding of the teeth and jerking of the head. The right ear appeared paralyzed and insensitive to mechanical stimulation. The rectal temperature was 106° F. Sacrificed for examination, the animal was found to be in excellent flesh. The prescapular and mesenteric lymph-glands had an edematous appearance, and the liver showed sharply defined yellowish lobules with dark brown centers. The histologic and bacteriologic findings corresponded in every respect to those of case 4.

Suggestive cases: Without a knowledge of cases 1 to 4, case 5 could have been mistaken for one of pregnancy disease. Another case showing the syndrome of pregnancy disease had been observed in the College flock during the fall of 1933, the diagnosis of which has since become somewhat problematic.

The following case is worthy of note. A two-month-old grade lamb was received from an outside flock in May, 1935. The animal exhibited symptoms which were suggestive of "white muscle lesions disease."[15] On pathologic and histologic examination, the voluntary muscles appeared normal; the cerebellum showed poly-

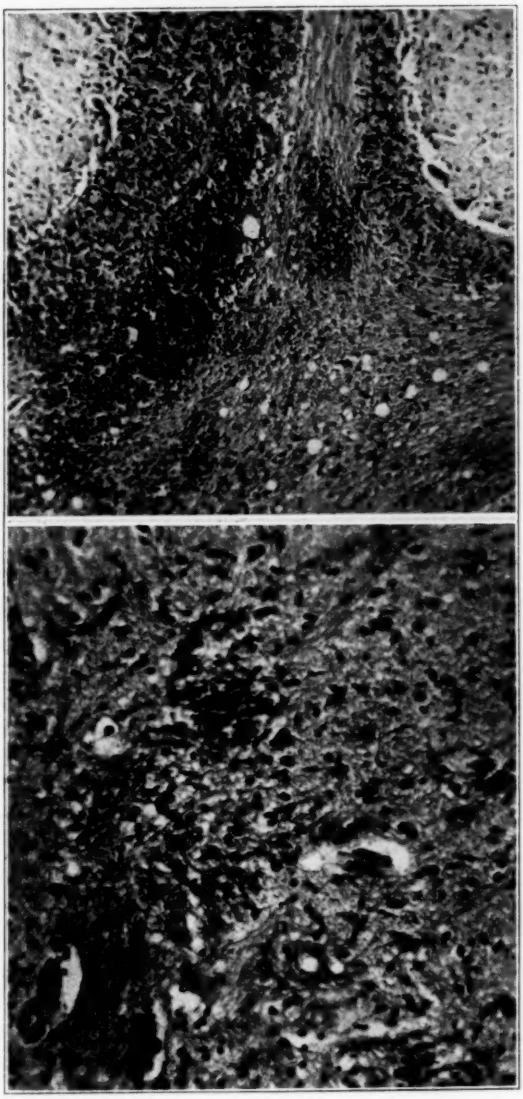


FIG. 5 (above). Cerebellum of non-specific encephalitis case in lamb. Note large polynuclear focus on inner aspect of left molecular layer and glia foci in center (x 70).

FIG. 6 (below). Medulla of mouse injected intranasally with sheep brain. Focus consists of polynuclear and mononuclear elements (x 100).

were diminished; the rectal temperature was 105° F., and the differential leukocyte count of the blood was within normal limits. When the animal appeared to be comatose, it was sacrificed for examination. The ventral cervical region exhibited a slight gelatino-hemorrhagic lesion in the subcutis, probably of traumatic origin. The abdominal cavity contained a small amount of transudate and the omasum and abomasum voluminous doughy ingesta. Fecal examination suggested moderate infestation with *Hemonchus contortus, Trichuris ovis, Moniezia sp.,* and coccidia. No evidence of albuminuria was obtained. Histologic examination of the internal organs was essentially negative, although the liver cells showed slight parenchymatous and hydropic degeneration and fatty metamorphosis. Medullary lesions of the granulocytic and monocytic type described in case 3 were marked and definite.

Since the granulocytic foci in the medulla of the previous case suggested a microbic origin of, or an association with, the disease, cultural studies of internal and cranial organs were conducted on a variety of media, including blood-agar slants and boiled egg-meat medium. Save for a few pigmented coccus colonies, the only significant growth was obtained from the medulla on the two media mentioned. This was identified later as a member of the genus *Listerella.*

Case 5: On January 11, 1936, a three-year-old Shropshire ewe in pen 2, known to be in an advance state of pregnancy, was found lying prone, with the head deflected to the left. When excited, the animal reacted by pawing, grinding of the teeth and jerking of the head. The right ear appeared paralyzed and insensitive to mechanical stimulation. The rectal temperature was 106° F. Sacrificed for examination, the animal was found to be in excellent flesh. The prescapular and mesenteric lymph-glands had an edematous appearance, and the liver showed sharply defined yellowish lobules with dark brown centers. The histologic and bacteriologic findings corresponded in every respect to those of case 4.

Suggestive cases: Without a knowledge of cases 1 to 4, case 5 could have been mistaken for one of pregnancy disease. Another case showing the syndrome of pregnancy disease had been observed in the College flock during the fall of 1933, the diagnosis of which has since become somewhat problematic.

The following case is worthy of note. A two-month-old grade lamb was received from an outside flock in May, 1935. The animal exhibited symptoms which were suggestive of "white muscle lesions disease."[15] On pathologic and histologic examination, the voluntary muscles appeared normal; the cerebellum showed poly-

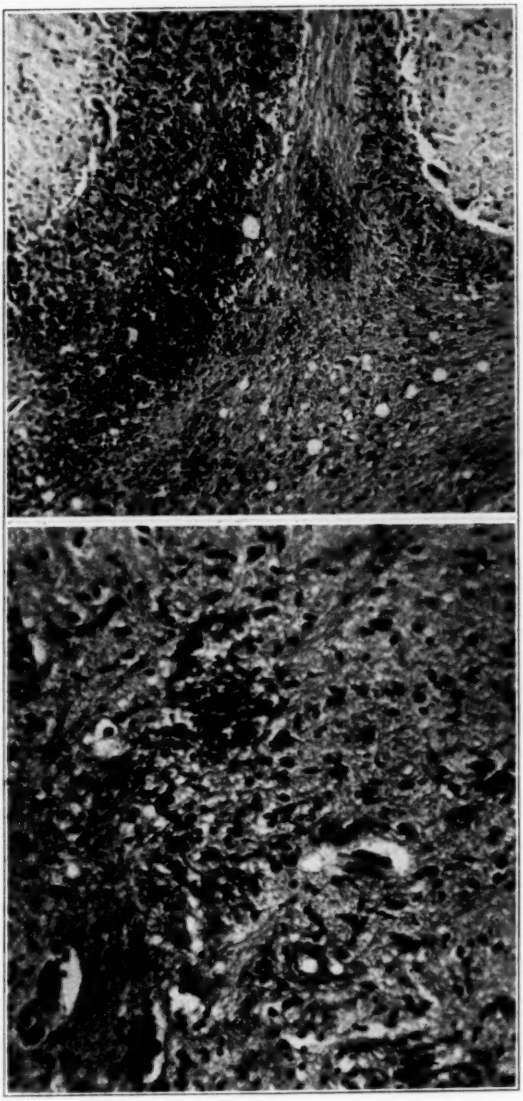


FIG. 5 (above). Cerebellum of non-specific encephalitis case in lamb. Note large polynuclear focus on inner aspect of left molecular layer and glia foci in center (x 70).

FIG. 6 (below). Medulla of mouse injected intranasally with sheep brain. Focus consists of polynuclear and mononuclear elements (x 100).

nuclear foci in and close to the granular layer (fig. 5), while the medulla exhibited patchy gliosis. On bacterioscopic and cultural examination of the affected organs, no significant organisms were found. As the umbilical arteries still contained soft and poorly organized thrombi, the cerebellar lesions were interpreted as metastatic lesions following primary omphalo-phlebitis. Failure to find *Listerella* organisms in this case should strengthen the conception of an epizoötiologic and etiologic entity in cases 1 to 5.

Transmission Experiments

Mice injected with fresh brain tissue: In a preliminary experiment, two white mice were injected intranasally, under light ether anesthesia, with a histologically typical glycerol-saline suspension of fresh medullary tissue. One of the mice experienced respiratory embarrassment on the 17th day following inoculation, when it was etherized. A Listerella-like organism was cultured from the medulla, and characteristic lesions were observed in sections of the medullary tissue (fig. 6) and of the posterior brain stem. Qualitatively, the lesions corresponded to those observed in field cases of sheep, although they were somewhat more extensive. The other mouse remained normal during a four-week period of observation, and proved to be susceptible to reinoculation with a *Listerella* culture by the same route. This experiment was repeated with material from another case, on ten mice of which five were injected with brain tissue and five with glycerol-saline only. Three of the brain-injected mice died in from 14 to 18 days and were found to be histologically positive, while the control mice, killed at the conclusion of the experiment, did not exhibit lesions suggestive of a brain affection (such as lymphocytic choriomeningitis).

Mice injected with filtered brain tissue: The medulla from another ovine case was stored in the refrigerator for a few hours, when frozen sections from it were shown to be typically affected. Part of this same tissue was used for the preparation of a 10 per cent suspension in hormone broth. The suspension was divided into four parts; one part was used for ordinary intranasal injection, while the three others were subjected to Berkefeld V, N, and Seitz E. K. filtration, respectively, and the filtrates injected by the same route. Five mice were used in each group. Three in the group which received the unfiltered material died from encephalitis; all of the other animals remained unaffected.

Intravenous inoculation of mice with culture: Brown and white mice which were injected intravenously with pure cultures

of *Listerella* isolated from spontaneous cases in sheep died within 24 hours. Control mice kept in the same cage did not contract the disease. At autopsy the inoculated animals presented the picture of septicemia which was characterized by tumefaction of the spleen and perilobular petechiae in the liver. The organism could be recovered from the internal organs, but not from the brain; it maintained its pathogenicity in serial tests.

Intranasal inoculation of mice with culture: Four out of five white mice which had been forced to snuff a small amount of culture suspension of *Listerella* under light ether anesthesia died within 48 hours. They presented the picture of patchy pneumonia. Tumefaction of the parenchymatous organs was only slightly pronounced, but brain involvement was suggested by injection of the meningeal vessels. The organism was recovered from the brain and internal organs.

In repeat experiments on another group of five mice, a very dilute suspension of the organism was used, with the result that only one out of five died after an incubation period of 14 days. This mouse showed brain lesions which were indistinguishable from those of mice which had received whole brain, while the mice which died after a short incubation period presented in a large measure the picture of acute hemorrhagic meningitis.

Inoculation of rabbits with culture: The first intravenous and intranasal injections of rabbits with 0.5 cc of a pure culture suspension of *Listerella* corresponding to tube 1 of the barium sulfate standard were innocuous. Later, the same strains administered in a similar dosage intravenously proved pathogenic, thus indicating an increase of virulence following a sojourn on laboratory media. The importance of dosage in transmission experiments with rabbits has been carefully worked out by Burn[13] and confirmed in these studies.

Inoculation of sheep with culture: Four eight-month-old grade Cheviot lambs secured from an outside flock and housed in the same pen were kept under general clinical observation for three days, at the end of which lamb 1 received a total of 2 cc of culture suspension (density of McFarland tube 2) intranasally, and lamb 2 a few drops of the same culture by the conjunctival route. Within 24 hours, the two lambs exhibited systemic symptoms of fever, and within five days they developed a liquid diarrhea, followed by loss of wool and ultimate recovery. Lamb 3 was kept as a temporary, and lamb 4 as a permanent, control. The pathogenicity of the organism for sheep, by the methods of inoculation used, is clearly shown in the accompanying temperature and agglutination charts (figs. 7 and 8). It will be noted that control

lamb 4 showed a decided reaction, which the author is inclined to interpret as being due to post-inoculative contagion, since escape of the culture fluid from nose or eye was prevented by restraint of the animal until absorption had taken place.

The development of subclinical infection may have been the explanation of some cases of transitory malaise which occurred in the College flock shortly after the actual outbreak of encephali-

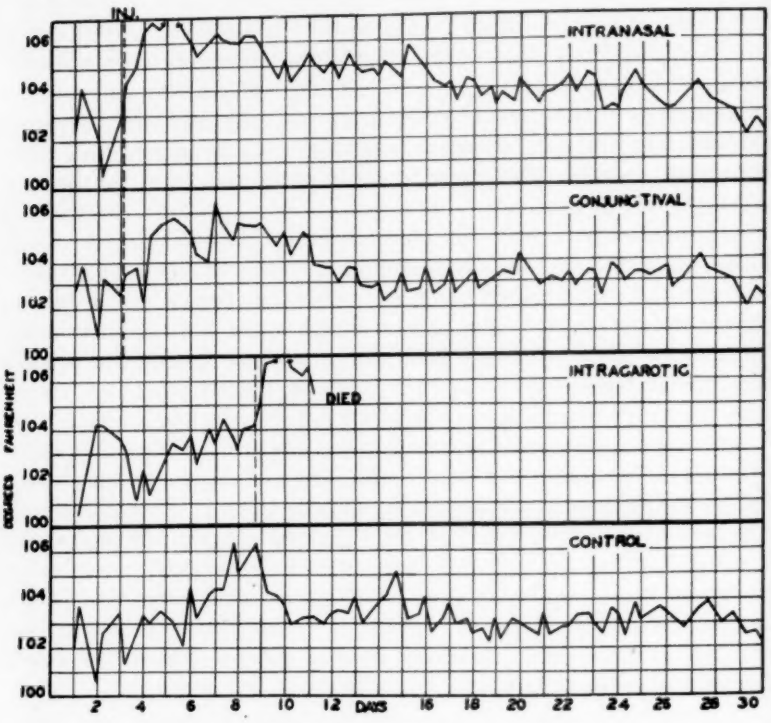


FIG. 7. Temperature charts of four sheep.

tis. Agglutination tests against *Listerella* conducted on ten animals from each of the five affected pens revealed nine animals with a titre of 1:25 or 1:50, but whether these reactions indicated a true carrier state or a temporary sensitization remains undecided, since this interesting line of inquiry could not be pursued at that time.

Lamb 3 was held under observation for nine days, and then injected intracarotically with 0.5 cc of culture suspension of McFarland density 1. Within 48 hours, the lamb lay prostrate on its side; the conjunctiva appeared severely congested, and the

rectal temperature was 106° F. The labored respiration was 80 p.m. of a superficial inspiratory type. The differential leukocyte count was within normal limits. The head was held almost rigidly retracted, and pawing movements were seen in the free extremities. Skin reflexes were wanting. Autopsy of the sacrificed animal revealed esophagostomiasis, and minute hemorrhages in the auricles and coronary fat of the heart. Opening of the cranial cavity brought to view severe hemorrhagic ecchymoses in the epidural and subdural spaces over the cerebellum and midbrain, and cloudy cerebrospinal fluid. A Listerella-like organism

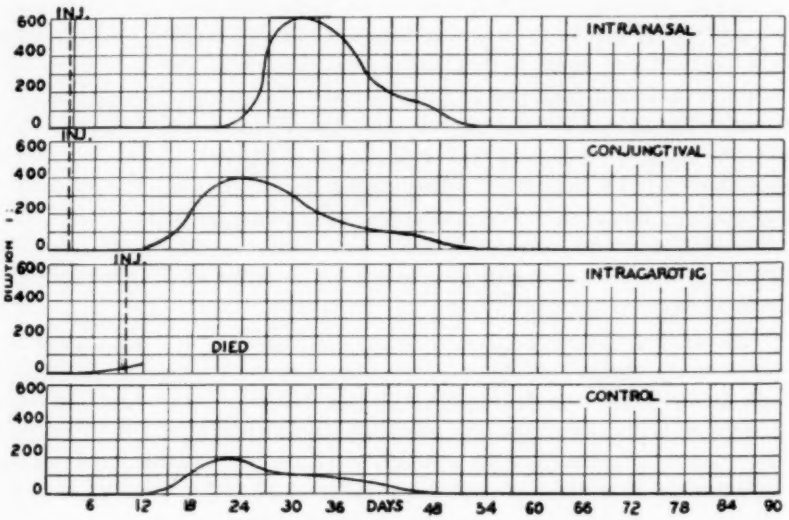


FIG. 8. Agglutination charts of four sheep.

was recovered from the medulla, cerebellum, corpora quadrigemina, cerebrum and cerebrospinal fluid, but not from the parenchymatous organs of the body cavities. The histologic lesions were characterized by hemorrhagic meningitis and inconspicuous monocytic reaction around the medullary vessels. The central polynuclear aggregates in the medulla, which were pathognomonic for spontaneous cases, could not be detected in the catastrophic experimental disease.

BACTERIOLOGIC STUDIES

As already stated, routine cultural examination of field cases failed to reveal an agent of etiological significance. Isolation of a Listerella-like organism from the medulla of cases 4 and 5 was effected on blood-agar slants and egg-meat medium. Visible growth on blood-agar slants appeared after incubation for 48

hours at 37° C. The two Storrs ovine strains were studied along with two human strains which were received from Dr. C. G. Burn, Yale University, and one ovine strain isolated by Dr. Ten Broeck, in 1930, and obtained through the courtesy of Dr. C. V. Seastone, Princeton, N. J.

Biological characteristics: The Storrs ovine strains were Gram-positive, non-spore-forming rods with rounded ends and having an average diameter of 1.7 x 0.7 μ (3.3 — .9 x 1.1 — .4 μ). The rods were usually seen to occur single or in pairs, very rarely in short chains, and differed from diphtheroid organisms in being quite uniform in shape and staining properties. In sections from field cases the organisms were confined to polynuclear foci in the medulla where they occurred in small groups the members of which tended to form acute angles, but not chains. Young laboratory cultures exhibited sluggish motility, which was lost before the end of a 24-hour period of incubation. Flagellar stains were inconclusive, due perhaps to the extreme fragility of the appendages, but suggested the presence of a single polar flagellum. Hemolytic power was not observed in primary cultures; on subculture, the organisms produced a narrow zone of beta hemolysis on salt-blood-agar plates.

Laboratory strains grew on meat infusion agar, but failed to develop on extract medium. Liver-agar proved to be the medium of choice from the standpoint of luxuriant growth and ease of colony recognition. On this medium the organism formed round, low, convex colonies with smooth surface and an entire border, 1-3 mm in diameter, which were distinguished by a milky-bluish, almost opalescent, appearance in transmitted light. Tests for ability to produce indol, hydrogen sulfide, acetyl methyl carbinol and reduction of nitrates were negative; the "methyl red" test was positive. Litmus milk was reduced in 24 hours, without coagulation; gelatin broth was not liquefied, but supported growth along the stab, generally in a beaded manner, although villous outgrowths occurred at times.

Fermentation reactions were studied in extract broth containing 1 per cent of the test substance and bromthymol blue as indicator, incubation being over a period of three weeks. Acid without gas was formed within 24 hours in broth containing dextrine, glucose, levulose, maltose, mannose and salicin; delayed acid formation (2-14 days) was observed in broth containing arabinose, galactose, glycerol, lactose, mannitol, raffinose, rhamnose, sorbitol, sucrose, trehalose and xylose; dulcitol and inulin were not attacked.

No outstanding cultural differences could be detected between the Storrs and the known stock strains. Minor differences occurred in "delayed" fermentation reactions; the three reference strains failed to attack galactose (contrary to Burn[13]); the two human strains attacked raffinose; sorbitol was fermented only by the reference strains. The Princeton strain did not attack lactose. Except in the case of mannitol, the minor differences seemed to divide the strains into Storrs ovine strains, on the one hand, and New Haven human and Princeton ovine strains, on the other. These results and previous reports in the literature would suggest that fermentation reactions are unsuitable for specific classification of organisms belonging to the genus *Listerella*.

Serologic studies: In several attempts to prepare antiserums by intravenous injection of rabbits with small doses of live culture, the animals succumbed after the third or fourth injection. Immunization was carried out, therefore, with fairly heavy doses of formalinized cultures over a period of three weeks. Three antiserums were prepared, one each against an ovine Storrs and a Princeton strain, and one against a New Haven human strain. The homologous titre of the serums varied between 1:800 and 1:1,600. In cross-agglutination tests the three serums agglutinated the five available antigens to the homologous titre limit, with the exception of one human strain which was practically unaffected by the Princeton ovine serum. Since the other human strain proved to be agglutinable by the same serum, this failure to cross-agglutinate was interpreted on the basis of antigenic deficiency of the exceptional human strain. Similar to cross-agglutination tests, reciprocal absorption tests did not show antigenic differences among the zoölogic varieties.

In a further extended study, an attempt was made to apply the principles of receptor analysis which have been eminently successful in the type-differentiation of *Salmonella*. Preparatory to this work the five available organisms were examined for evidence of bacterial variation, with the result that, in spite of S colony formation, the strains behaved like R variants in the acriflavine and boiling tests. The R tendency made it impossible to prepare alcohol-extracted O-antigens. Repeated platings and subsequent pickings of colonies did not result in the establishment of strains negative to the acriflavine test. The results with O-antigens prepared from live, non-motile variants did not differ from those obtained in ordinary agglutination and mirror absorption tests. The H-titre of both anti-ovine strain serums was about 1:100, which made comparative studies inadvisable.

Summary

Five cases of meningo-encephalitis associated with a Listerella-like organism in a flock of 200 pure-bred sheep are reported here. The first recognized case occurred approximately one year before a small epizoötic affecting four animals made its appearance. The diagnosis was based in one case on clinical observation alone, in two instances on histologic and bacterioscopic evidence, and in two animals on histologic, bacterioscopic and cultural findings. Similar cases in sheep have been reported by Seastone in the United States, and by Gill under the name of "circling disease" in New Zealand.

Pathologically the disease was characterized by affecting well-nourished mature animals of both sexes, by normal differential leukocyte count of the blood, slight parenchymatous and fatty changes in the liver, and centrally located polynuclear foci and monocytic perivascular and meningeal infiltration in the medulla oblongata. Disturbances similar to those observed in the sheep were produced in mice by intranasal injection of brain suspensions and of small doses of culture after an incubation period of about two weeks. Filtered brain material was innocuous, while large doses of culture administered by the same route produced a rapidly fatal septicemia and meningitis. Sheep infected with the cultured organism by intranasal or conjunctival application showed a marked thermic and agglutinative response, and recovered; a similar but slighter reaction was observed in a control animal kept in the same pen. Intracarotid injection of sheep caused death from hemorrhagic meningitis within 48 hours.

Two ovine *Listerella* strains isolated from the outbreak were found to be indistinguishable by cultural, agglutination, and agglutinin-absorption tests from one ovine and two human stock strains. Although the pathogenesis of the ovine disease is not entirely understood, it appears that the *Listerella* genus plays an important rôle as an etiological agent.

References

[1]Murray, E. G. D., Webb, R. A., and Swann, M. B. R.: A disease of rabbits characterized by a large mononuclear leucocytosis, caused by a hitherto undescribed bacillus, *Bacterium monocytogenes* (n. sp.). Jour. Path. & Bact., xxix (1926), pp. 407-439.

[2]Pirie, J. H. H.: A new disease of veld rodents. "Tiger River Disease." So. African Inst. Med. Res., iii (1927), pp. 163-186.

[3]Bergey, D. H.: Manual of Determinative Bacteriology. (4th ed., William & Wilkins Co., Baltimore, 1934), p. 328.

[4]Nyfeldt, A.: Etiologie de la mononucléose infectieuse. Compt. Rend. Soc. Biol., ci (1929), pp. 590-592.

[5]Anton, W.: Kritisch-experimenteller Beitrag zur Biologie des *Bakterium monocytogenes*. Mit besonderer Beruecksichtigung seiner Beziehung zur infektioesen Mononukleose des Menschen. Cent. f. Bakt., Abt. I, Orig. Bd. cxxxi (1934), pp. 89-103.

[6]Olitsky, P. K., Syverton, J. T., and Tyler, J. R.: Studies on the etiology of spontaneous conjunctival folliculosis of rabbits. Jour. Exp. Med., lx (1934), pp. 375-384.
[7]Gill, D. A.: "Circling" disease of sheep in New Zealand. Vet. Jour., lxxxvii (1931), pp. 60-74.
[8]Gill, D. A.: Circling disease, a meningo-encephalitis of sheep in New Zealand. Vet. Jour., lxxxix (1933), pp. 258-270.
[9]Jones, F. S., and Little, R. B.: Sporadic encephalitis in cows. Arch. Path., xviii (1934), pp. 580-581.
[10]Schultz, E. W., Terry, M. C., Brice, A. T. Jr., and Gebhardt, L. P.: Bacteriological observations on a case of meningo-encephalitis. Proc. Soc. Exp. Biol. & Med., xxxi (1934), pp. 1021-1023.
[11]Seastone, C. V.: Pathogenic organisms of the genus *Listerella*. Jour. Exp. Med., lxii (1935), pp. 203-212.
[12]Burn, C. G.: Unidentified Gram-positive bacillus associated with meningo-encephalitis. Proc. Soc. Exp. Biol. & Med., xxxi (1934), pp. 1095-1097.
[13]Burn, C. G.: Clinical and pathological features of an infection caused by a new pathogen of the genus *Listerella*. Amer. Jour. Path., xii (1936), pp. 341-348.
[14]Burn, C. G.: Characteristics of a new species of the genus *Listerella* obtained from human sources. Jour. Bact., xxx (1935), pp. 573-591.
[15]Jungherr, E., and Welch, H.: A report on lamb diseases. Jour. A. V. M. A., lxxii (1927), n. s. 25 (3), pp. 317-326.

Dogs in March of Time

A picture titled, "Dogs for Sale," arranged by the weekly news magazine *Time* on the "March of Time" program, was shown recently at leading theaters throughout the country. The movie included hospitalization of dogs, catering service for dogs, stores where dog supplies are sold, dog cemeteries, and other features. The picture also showed dogs in training at "The Seeing Eye," the world-famous institution at Morristown, N. J., where dogs are trained to guide the blind. One scene was taken in the office of Governor Lehman, of New York, showing him signing a bill, recognizing as one individual the blind master and his "seeing dog." Scenes of a congressional committee, in session in Washington, listening to the plea of a blind doctor asking for legislation to broaden the privileges of blind people and their dogs on public conveyances, also were shown.

Coronation Edition

In keeping with the preparations for the coronation of King George VI, of England, the May number of *The Veterinary Journal* (London) was dressed in an attractive gold cover. The coronation edition included photographs of the King, on his favorite mount, and the Windsor Greys which drew the coronation coach. The harness for each of the eight horses is of red morocco leather with ornamentations of gold plate and copper and has no "joins." Each side of a harness is made from a single hide and is so heavy that two men are necessary to lift it into position on the horse.

LOSSES OF UNDETERMINED CAUSE FOLLOWING AN OUTBREAK OF EQUINE ENCEPHALOMYELITIS*

By HADLEIGH MARSH, *Helena, Mont.*
Montana Livestock Sanitary Board

In the summer of 1936, there were reported 322 cases of equine encephalomyelitis in Montana. Eighteen counties were involved in the outbreak, but 275 of the cases were in three areas, involving eight counties. The first case was reported on July 25, in Ravalli County, where 96 of the cases occurred. The last case reported in Ravalli County was on September 5, but some cases occurred in other areas until about October 1. The laboratory diagnosis was made by Dr. H. R. Cox, of the Rocky Mountain Laboratory of the United States Public Health Service, at Hamilton. Dr. Cox recovered the virus from two horses early in the outbreak and determined that it was of the western type.

When the disease appeared in western Montana, the question arose as to what methods of control to adopt. As quarantine measures had been found in other states to be impracticable, and treatment was at the best uncertain, the use of immunizing agents remained as a possibility. Formolized vaccine had been shown to be of value in producing an immunity of about eight months duration, but the use of this product alone seemed impractical after the start of an outbreak, as it is known that the immunity is not fully developed until about three weeks after the first dose is given.

The use of anti-encephalomyelitis serum is known to produce a temporary passive immunity, but after about two weeks, the treated horses are again susceptible. The combined use of serum and formolized vaccine should be effective for one season, but it is an expensive procedure, when one takes into consideration that as a rule only a few cases occur on a ranch, and at most 10 per cent of the horses in an area are attacked. It was decided not to recommend general immunization, but if certain owners desired to attempt immunization of their horses, to recommend the use of serum and virus simultaneously, as described by Records and Vawter,[1] in Nevada.

Virus of the Nevada strain which had been used by Records and Vawter was obtained through the courtesy of Dr. Records, and the Montana Veterinary Research Laboratory prepared virus

*Received for publication, May 7, 1937.

for use by representatives of the Livestock Sanitary Board in the field. This virus was prepared and distributed in accordance with the recommendations of Records. Guinea pigs were inoculated intracranially with 0.1 cc of a 1-100 dilution of a 20 per cent suspension of virulent guinea pig-brain tissue. On the third or fourth day after inoculation, the guinea pigs were killed. The brain was removed aseptically and ground in a sterile mortar, and sufficient 50 per cent buffered glycerin of pH 7.4-7.5 added to make a 20 per cent suspension. This suspension was poured through gauze into centrifuge-tubes and centrifuged at a low speed for ten minutes to throw out the coarser brain particles. The 20 per cent suspension was distributed in 1-cc quantities in small vials. Each 1-cc vial was shipped with 9 cc of buffered saline in a separate container. At the time of use, the 1 cc of virus was mixed with the 9 cc of saline solution. The dose was 2 cc of the mixture for adult horses and 1 cc for colts under one year old. The virus was kept refrigerated at all times and shipped in iced containers. The virus was injected subcutaneously and, at the same time, 50 cc of serum was injected.

TABLE I—*Statistics on equine encephalomyelitis in* 1936.

County	Cases	Horses Involved	Ranches Involved	Horses Immunized	
				Serum-Virus	Serum Alone
Ravalli	96	737	74	2109	0
Missoula	38	450	34	333	0
Lake	39	99	34	540	0
Cascade	31	157	27	410	0
Pondera	18	134	16	354	0
Teton	16	98	14	307	0
Yellowstone	21	124	21	77	28+
Carbon	16	87	11	0	73
Madison	10	67	7	142	34
Beaverhead	7	23	5	0	16
Lewis and Clark	1	5	1	60	0
Broadwater	1	8	1	0	0
Judith Basin	10	15	2	0	5
Wheatland	1	8	1	0	0
Hill	2	14	2	0	0
Blaine	6	68	6	0	0
Richland	1	8	1	0	0
Rosebud	8	19	1	0	0
Forest service in western Montana	0	0	0	861	0
Totals	322	2121	258	5193	156+

LOSSES OF UNDETERMINED CAUSE FOLLOWING AN OUTBREAK OF EQUINE ENCEPHALOMYELITIS*

By HADLEIGH MARSH, *Helena, Mont.*
Montana Livestock Sanitary Board

In the summer of 1936, there were reported 322 cases of equine encephalomyelitis in Montana. Eighteen counties were involved in the outbreak, but 275 of the cases were in three areas, involving eight counties. The first case was reported on July 25, in Ravalli County, where 96 of the cases occurred. The last case reported in Ravalli County was on September 5, but some cases occurred in other areas until about October 1. The laboratory diagnosis was made by Dr. H. R. Cox, of the Rocky Mountain Laboratory of the United States Public Health Service, at Hamilton. Dr. Cox recovered the virus from two horses early in the outbreak and determined that it was of the western type.

When the disease appeared in western Montana, the question arose as to what methods of control to adopt. As quarantine measures had been found in other states to be impracticable, and treatment was at the best uncertain, the use of immunizing agents remained as a possibility. Formolized vaccine had been shown to be of value in producing an immunity of about eight months duration, but the use of this product alone seemed impractical after the start of an outbreak, as it is known that the immunity is not fully developed until about three weeks after the first dose is given.

The use of anti-encephalomyelitis serum is known to produce a temporary passive immunity, but after about two weeks, the treated horses are again susceptible. The combined use of serum and formolized vaccine should be effective for one season, but it is an expensive procedure, when one takes into consideration that as a rule only a few cases occur on a ranch, and at most 10 per cent of the horses in an area are attacked. It was decided not to recommend general immunization, but if certain owners desired to attempt immunization of their horses, to recommend the use of serum and virus simultaneously, as described by Records and Vawter,[1] in Nevada.

Virus of the Nevada strain which had been used by Records and Vawter was obtained through the courtesy of Dr. Records, and the Montana Veterinary Research Laboratory prepared virus

*Received for publication, May 7, 1937.

for use by representatives of the Livestock Sanitary Board in the field. This virus was prepared and distributed in accordance with the recommendations of Records. Guinea pigs were inoculated intracranially with 0.1 cc of a 1-100 dilution of a 20 per cent suspension of virulent guinea pig-brain tissue. On the third or fourth day after inoculation, the guinea pigs were killed. The brain was removed aseptically and ground in a sterile mortar, and sufficient 50 per cent buffered glycerin of pH 7.4-7.5 added to make a 20 per cent suspension. This suspension was poured through gauze into centrifuge-tubes and centrifuged at a low speed for ten minutes to throw out the coarser brain particles. The 20 per cent suspension was distributed in 1-cc quantities in small vials. Each 1-cc vial was shipped with 9 cc of buffered saline in a separate container. At the time of use, the 1 cc of virus was mixed with the 9 cc of saline solution. The dose was 2 cc of the mixture for adult horses and 1 cc for colts under one year old. The virus was kept refrigerated at all times and shipped in iced containers. The virus was injected subcutaneously and, at the same time, 50 cc of serum was injected.

TABLE I—*Statistics on equine encephalomyelitis in* 1936.

County	Cases	Horses Involved	Ranches Involved	Horses Immunized	
				Serum-Virus	Serum Alone
Ravalli	96	737	74	2109	0
Missoula	38	450	34	333	0
Lake	39	99	34	540	0
Cascade	31	157	27	410	0
Pondera	18	134	16	354	0
Teton	16	98	14	307	0
Yellowstone	21	124	21	77	28+
Carbon	16	87	11	0	73
Madison	10	67	7	142	34
Beaverhead	7	23	5	0	16
Lewis and Clark	1	5	1	60	0
Broadwater	1	8	1	0	0
Judith Basin	10	15	2	0	5
Wheatland	1	8	1	0	0
Hill	2	14	2	0	0
Blaine	6	68	6	0	0
Richland	1	8	1	0	0
Rosebud	8	19	1	0	0
Forest service in western Montana	0	0	0	861	0
Totals	322	2121	258	5193	156+

While it was originally intended to use this method on only a small number of horses, there was an unexpected demand for immunization, and the result was that 5,193 horses were immunized by this method. All the vaccinated horses were on ranches where the disease occurred, or in the immediate vicinity. Table I shows distribution of the disease by counties and the number of horses immunized in each county.

The results of this work indicate that immunity was immediately established. In the vaccinated horses, five cases of encephalomyelitis occurred within four days following vaccination, which indicates that the disease was already developing before vaccination. Four cases were reported as occurring between five and 19 days after vaccination, but the diagnosis is questionable in these cases. On the other hand, on one of the ranches where no immunizing agent was used, several cases occurred over a period of two months following the first case. The question of whether a permanent immunity was established cannot yet be answered.

Outbreak Following Immunization

Between October 2 and November 16, there occurred an outbreak of disease in horses in the areas where encephalomyelitis occurred in August, the nature of which is as yet unexplained. This group of cases corresponded very closely to the "second wave" of encephalomyelitis described by Madsen,[2] in Utah, in 1933. Similar outbreaks were reported in Colorado and Wyoming in 1936.

Table II—*Statistics of horse disease following encephalomyelitis.*

County	Cases	Cases on Previously Infected Ranches	Immunization History			Dates
			Serum-Virus	Serum Alone	Not Immunized	
Ravalli	51	7	45	0	6	10/2 –11/16
Missoula	8	0	6	0	2	10/27–11/11
Lake	5	0	5	0	0	10/28–11/2
Sanders	1	0	1	0	0	11/9
Cascade	14	10	14	0	0	10/12–11/8
Pondera	3	3	3	0	0	10/7 –11/10
Teton	1	1	1	0	0	10/20
Carbon	5	2	0	5	0	10/14–11/6
Beaverhead	1	1	0	1	0	10/9
Totals	89	24	75	6	8	

There were 89 cases of this disease, with 79 fatalities. Table II shows their distribution by counties. Eighty-one of the 89 cases were in horses which had been immunized during the August outbreak of encephalomyelitis. The time interval between the date of vaccination and the appearance of the secondary disease varied from 32 to 92 days, 90 per cent of the cases occurring between 40 and 70 days after immunization.

The clinical symptoms in these cases differed very definitely from the symptoms observed in the encephalomyelitis cases in August. The disease was very acute, and was fatal in 90 per cent of the cases, as compared with about 40 per cent mortality in the summer cases. In many cases the horses lived only 24 to 48 hours after symptoms were first observed. The symptoms were those of excitement, as contrasted with the sleepy symptoms of typical encephalomyelitis. The horses would not stand quietly. They walked continually, and attempted to push through any obstacles, such as fences, resulting in severe bruises and lacerations. There were muscular tremors, impaired vision, and profuse sweating. In some cases the mouth was held open. The mucous membranes were somewhat icteric. No rise in temperature was observed at any time in any of the horses in the Bitter Root Valley, where quite extensive observations were made. The pulse remained normal, and the respiration was practically normal until the last stages of the disease. In most of these horses there was no prostration. They died on their feet.

Postmortem Findings

Autopsies were made on a number of the horses, and certain lesions were found in all cases. The stomach was always distended with rather dry food material. The mucosa of the stomach was hyperemic. A catarrhal enteritis was characteristic, with more or less hyperemia of the mucosa of the entire intestinal tract. The liver was enlarged, showing the gross appearance of chronic passive congestion. Fibrinous shreds were always found on the diaphragmatic surface of the liver. The spleen was doughy in consistency, and the kidneys showed some parenchymatous degeneration, with petechiae in the cortex. There was no gross pathological change in the brain and spinal cord.

Tissues from four horses were used for inoculation of guinea pigs, rabbits and mice. Intracranial inoculations of material from the brain stem, cerebrum, cerebellum, hippocampus, lumbar cord, and spinal fluid were made into 37 guinea pigs, two rabbits and two mice. Citrated blood of one of the horses was inoculated into two guinea pigs and two rabbits. The results of these

inoculations were all negative for encephalomyelitis virus. Dr. Cox, of the United States Public Health Service Laboratory, also failed to demonstrate the presence of encephalomyelitis virus in tissues from these horses.

In view of the constant lesions in the stomach and intestines, a Berkefeld filtrate was prepared from a suspension of tissue from the mucosa of the stomach, duodenum and colon of one horse. This filtrate was inoculated intracranially, intraperitoneally and subcutaneously into guinea pigs, with negative results.

Histological examination was made of the cerebrum, hippocampus and lumbar cord of one horse, and the cerebrum, cerebellum, hippocampus and lumbar cord of another horse. No lesions could be recognized in any of the sections.

Histological examination of the liver of two horses showed degeneration of the cytoplasm of the liver cells around the efferent vein, with pigment deposits in the degenerated cells.

Cause of Losses Undetermined

The cause of these losses following the encephalomyelitis outbreak is as yet undetermined. The attempts of the two laboratories to demonstrate the presence of the virus of equine encephalomyelitis in these cases have had uniformly negative results. While there was clinical evidence of acute cerebral disturbance, the symptoms in these cases differed very definitely from those observed in the encephalomyelitis outbreak. A limited amount of histological work has failed to establish the presence of lesions in the central nervous system.

When the first cases appeared in horses which had been immunized with serum and virus, our first thought was that this was a delayed result of the virus injected. It was soon evident that this was not the case, when similar cases appeared in another part of the state in horses which had been immunized with serum alone. The occurrence of similar cases in Colorado and Wyoming, where no virus was used, proves that the virus was not responsible.

The fact that of the 89 Montana cases, 81 occurred in horses which had been immunized either with serum and virus or serum alone, and that no cases occurred outside of the original areas where encephalomyelitis existed, seems to justify a suspicion that the serum injection was in some way connected with the later losses, although the number of cases reported constituted only 1.5 per cent of the total number to which serum had been administered. Although eight cases were reported in horses which

had never been immunized, none of these cases developed until after a number of vaccinated horses in the same area had succumbed.

A group of 861 horses which were immunized with serum and virus constituted evidence that the serum injected was not directly responsible for the later losses. These horses were scattered over the whole of the western part of the state, but the only two authenticated cases of the secondary disease which occurred in this group of horses were in Missoula County, in the area where encephalomyelitis existed in August. Most of the 861 horses were in the mountain areas at some distance from the infected ranch areas, and no cases occurred in such horses.

While it is apparent that neither the serum nor virus was in itself responsible for later losses, it is difficult to dismiss the suspicion that the use of serum in infected areas was in some way connected with these losses. One large ranch in the Bitter Root Valley immunized 127 horses in August. Before vaccination, there had been one case of encephalomyelitis in these horses. In October, this outfit lost six horses with the secondary disease, only one of which had not been vaccinated in August. Another instance which is difficult to explain is that of a man who had only two horses, both of which were immunized in August. Only a few horses in the immediate vicinity had been immunized. In October, this man lost both these horses with the secondary disease which we have described.

It should be stated here that if there is any indirect relation between the use of serum and the development of the secondary disease which we have described, there is no implication of any responsibility on the part of the producers of the serum. The serum used in Montana was obtained from several commercial laboratories.

Conclusions

We must conclude that the acute and fatal disease which occurred following the 1936 epidemic of encephalomyelitis in Montana was probably not simply a second outbreak of the disease in horses in which no permanent immunity had been established. It is doubtful whether this secondary disease can be considered as encephalomyelitis, as it has been impossible to recover the virus from these cases or to demonstrate the lesions in the central nervous system.

References

[1]Records, E., and Vawter, L. R.: Equine encephalomyelitis immunization. Jour. A. V. M. A., lxxxiv (1934), n. s. 37 (5), pp. 784-798.

[2]Madsen, D. E.: Equine encephalomyelitis. Utah Acad. Sci., Arts & Letters, xi (1934), pp. 95-99.

PARASITIC TUMORS IN WILD BIRDS*

By L. C. MORLEY *and* J. E. SHILLINGER

Bureau of Biological Survey, U. S. Department of Agriculture
Washington, D. C.

The description of *Harpyrynchus longipilus* Banks,[1] in 1906, from a tumor under the wing of a crossbill, appears to be the earliest record of the "tumor-forming" mite in the United States. The rare occurrence of this parasite in the birds of this continent is revealed by an examination of the literature. The files of the National Museum record three cases affected with mites of the genus *Harpyrynchus;* two specimens of Wilson's thrush collected in May, 1907, from Ontario, and an eastern song sparrow from Virginia, in 1923. Tumor formation on an evening grossbeak by the mite *Harpyrynchus brevis* (Ewing) was reported in 1911.[2]

A female red-winged blackbird, collected in New York State, affected with an extensive tumorous growth (fig. 1), was received in the laboratory of the Division of Wildlife Research, Bureau of Biological Survey, in October, 1934. The surface was roughened and nodular in appearance, and covered by a firm crust. This growth, yellowish orange in color, was dry and crumbly in consistency, and covered the breast, the underpart of the neck, and parts of the thighs, and extended up under the wings onto the back. Numerous mites, identified by Dr. H. E. Ewing, of the Bureau of Entomology and Plant Quarantine, as *Harpyrynchus brevis* (fig. 2), were found on examination of the surface material, while the deeper portions contained many eggs and a few mites.

The description of the mite *Harpyrynchus brevis,* as given by Ewing, is as follows:

> General appearance hyaline. Integument finely striated by concentric lines for a large zone next to the margins of the body.

*Received for publication, May 15, 1937.

FIG. 1. Parasitic tumors of red-winged blackbird. (B-5509-M)

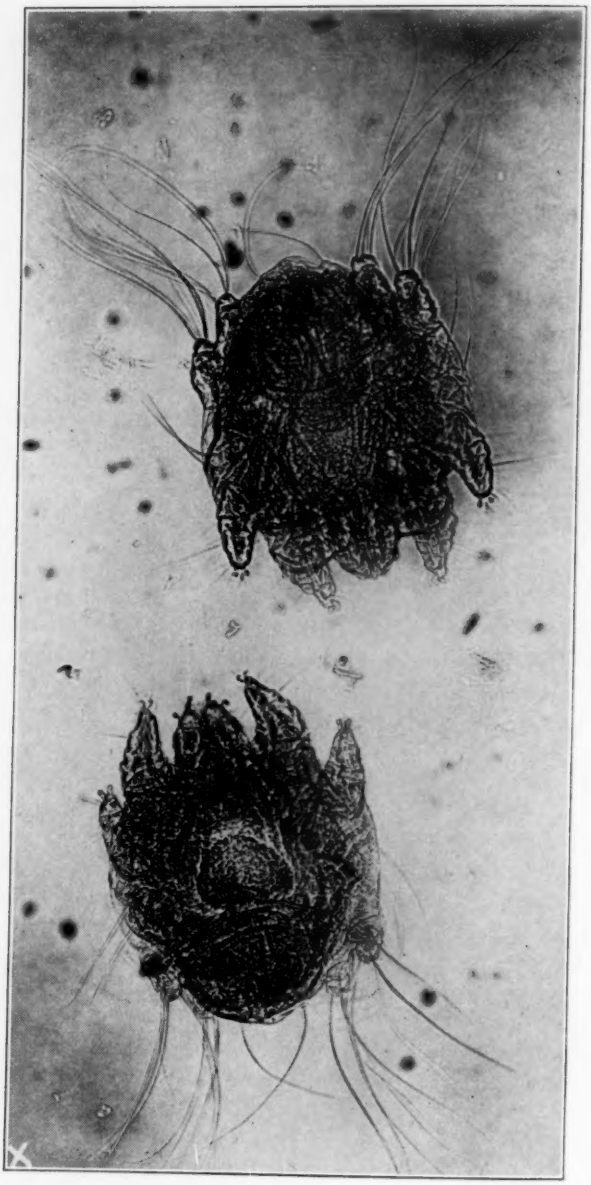


FIG. 2. Tumor-forming mite, *Harpyrynchus brevis*.
(B-6178-M)

This striated area extends inward for about one-half the distance from the margin to the center of the body.

Beak large, stout, extending forward to about the tip of the first pair of legs. Dorsal hooks four in number, stout and strongly recurved. Each palpus bears a very long bristle about equal to the total length of the beak.

Body circular in outline with sinuate margins. On the dorsal surface near the base of the beak there is a large pair of posteriorly directed bristles which extend almost to the posterior margin of the body; a similar but slightly longer pair of lateral bristles is present just behind the second pair of legs.

Anterior group of legs stout, subequal, and terminated each with two claws; tibia and tarsus subequal in length, the former somewhat broader than the latter. Posterior group of legs very short and each terminated with from five to seven enormous bristles which are longer than the body of the mite. The last pair of legs extends to the margin of the abdomen.

Length, 0.28 mm., breadth, 0.24 mm.

The feet of the red-winged blackbird also appeared to be affected with mange. Mange of the feet caused by a mite of the genus *Cnemidocoptes* is not of uncommon occurrence in wild birds, but no mites of any species were found on examination of the feet of this specimen.

REFERENCES

[1]Banks, N.: Descriptions of some new mites. Proc. Ent. Soc. of Wash., vii (1905), no. 2-3, p. 135.

[2]Ewing, H. E.: New predaceous and parasitic acarina. Psyche, xviii, p. 38.

ADENOCARCINOMA IN MINK*

By EARL F. GRAVES, *Poynette, Wis.*
Wisconsin State Fur Farm

A mature female mink was presented for autopsy, with little history. She had died suddenly and about 50 days before her death she had accepted the male's services. She was in good physical condition and had eaten normally.

A few weeks after she had mated, the owner noticed that she had begun to swell in the abdominal region and naturally supposed that she was pregnant.

Autopsy showed the only pathology to be located in the body of the uterus. Both ovaries and one horn were normal. There was a small growth on one horn, but the body had lost all definition and was an enlarged mass of tissue weighing 131 grams (fig. 1).

There was no hemorrhage into the abdominal cavity. My diagnosis to the owner was malignant carcinoma. Dr. L. McGary,

*Received for publication, May 25, 1937.

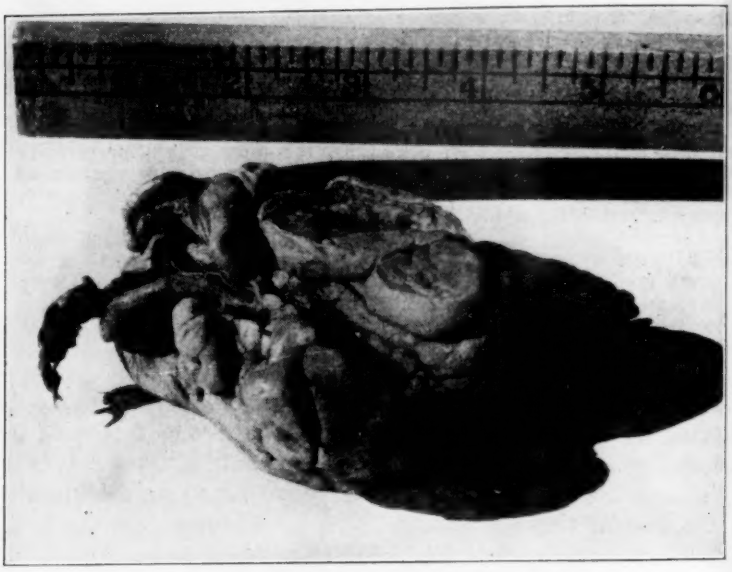


FIG. 1. Adenocarcinoma in mink.

pathologist at Madison General Hospital, Madison, Wisconsin, kindly ran a bit of the tissue through his permanent sectioning setup and reported back: adenocarcinoma.

ANOTHER METHOD OF CONTROLLING HEMORRHAGE IN DEHORNING*

By P. L. ROUSE, *Erie, Pa.*

In the June issue of the JOURNAL, Dr. John F. Ryff described a new ligature for use in dehorning. No doubt this would be a very satisfactory method to control the hemorrhage in such cases.

Another method which I have used with good results is as follows: After the horn has been removed, grasp the protruding end of the artery with a pair of hemostatic forceps and pull out as much of the artery as possible. Often about an inch will come away, which allows the blood to coagulate within a short time. There is no danger of any hemorrhage developing at a later time if the bleeding is stopped at the time of the operation. It is easier to grasp the artery if the horn is cut close to the head.

I hope that this suggestion may be helpful to other veterinarians.

*Received for publication, June 15, 1937.

THE USE OF CUTANEOUS STAPHYLOCOCCUS LESIONS IN MICE FOR THE EVALUATION OF THE GERMICIDAL ACTIVITY OF DISINFECTANTS. George A. Hunt. Jour. Inf. Dis., lx (1937), p. 232.

Skin lesions were produced in mice using an "invasive" strain *of Staphylococcus aureus.* The germicide was administered at the site of injection at once or subsequent to infection by the microörganisms and prevention of the formation of the lesion determined by comparison with control mice receiving an infection without germicide. Of the disinfectants tested, none prevented the formation of skin lesions when administered later than two hours after infection. None of the disinfectants was able to shorten the time of healing once the infection was established at the site of injection. The disinfectants of the alkylphenol and alkyl-resorcinol types and their halogen substitution products were able to prevent the formation of skin lesions in most instances when administered at the site of infection not more than one hour following infection by the organisms. The organic mercury compounds were found to be slow in action and much of their activity reversed by washing the cells with saline or by washing and treating with hydrogen sulfide.

BRUCELLA DISEASE IN WHITE MICE. Charlotte H. Singer-Brooks. Jour. Inf. Dis., lx (1937), p. 267.

Normal white mice succumb quickly to large doses of living Brucella organisms (300,000,000 to 400,000,000). Normal mice subjected to smaller doses (30,000,000 to 300,000 organisms) manifest no symptoms of infection but develop necrotic areas in the liver and enlargement of the spleen. Infections of 100,000 to 1,000 organisms do not produce gross anatomical changes but the organisms are harbored in various tissues for a long period of time. White mice immunized with heat-killed *Brucella suis* vaccine survive intraperitoneal injection of 400,000,000 living *Br. suis* organisms, which is fatal to control mice. Pregnancies in immunized mice, subjected to dosage of living *Br. suis* which was fatal to control mice, were not terminated by abortion. The

young born one to two weeks after the injection not only survived but developed into healthy adult mice. Immunized mice subjected to small infections do not manifest any appreciable increase in ability to free their tissues of Brucella organisms as compared with control mice.

AN INTERPRETATION OF THE VIRULENCE OF TUBERCLE BACILLI BASED ON EXPERIMENTAL OBSERVATIONS: MYCOBACTERIUM NUSQUAM PHYMATIOSIS. H. J. Corper. Jour. Inf. Dis., lx (1937), p. 312.

An avirulent strain of tubercle bacilli is defined as a strain of tubercle bacilli lacking the ability to multiply in a highly susceptible host when injected intravenously in a fine suspension in amounts approximating the threshold of tubercle formation (0.01 to 0.001 mg produces a local tubercle), as is evident by the lack of gross tuberculosis, or of the development of metastatic gross tuberculosis following the subcutaneous injection of relatively large amounts of a fine suspension of the bacilli (approximating 10 to as much as 500 mg). Three strains of human or bovine avirulent tubercle bacilli were studied. The property and consistency of avirulence and virulence of human and bovine tubercle bacilli are discussed. It is suggested that since "*Mycobacterium tuberculosum*" refers to a pathogene capable of producing tuberculosis, the avirulent strains should not be designated as such but as "*Mycobacterium nusquam phymatiosis*," rather than by names or synonyms such as BCG, R1, etc.

BOVINE MASTITIS IN RELATION TO MILK-BORNE EPIDEMICS. David J. Davis. Jour. Inf. Dis., lx (1937), p. 374.

The increase of milk-borne epidemics of disease traced to infected udders and the emphasis of veterinarians on the increasing frequency of mastitis in cows indicate the importance of intensive studies of mastitis in all its relations. Hemolytic streptococci applied to the teat surface will readily ascend the teat-ducts to the milk-cisterns, resulting in an infection. Similar experiments using strains of staphylococci have given negative results. However, when staphylococci of human origin are injected into the lactiferous duct a short distance above the meatus, the infection will "take," resulting in a definite and easily recognized mastitis. The rôle of infections, the anatomy and the histology of the teat-ducts, as relating to the problem, and the pathology of the udder as a focus of disease, are discussed.

VARIATIONS IN FOWL-PEST VIRUS. R. D. McKenzie and G. F. Findlay. Brit. Jour. Exp. Path., xviii (1937), p. 138.

Intracerebral passage in passively immunized canaries has resulted in the production of a variant of the fowl-pest virus. A second variant was produced by passaging the strain obtained from the canary intracerebrally in mice. The latter, it was found, could easily be infected with the canary variant, although they were only slightly susceptible to the action of the original or starting strain. The second or mouse variant differed from the canary variant and from the original strain in being even more highly pathogenic for mice. The varying degrees of pathogenicity in mice provided a ready means of distinguishing between these three strains. The two variants and the original strain are antigenically related.

THE TRANSMISSION OF FOWL-PEST TO FERRETS. G. M. Findlay and R. D. McKenzie. Brit. Jour. Exp. Path., xviii (1937), p. 146.

The ferret and the fitch are highly susceptible to the virus of fowl-pest. One strain of fowl-pest virus killed ferrets on intracerebral and intraperitoneal inoculation as well as on intranasal instillation; a second failed to kill on intraperitoneal inoculation but killed after intracerebral inoculation; another strain failed to kill but produced a febrile reaction on intracerebral inoculation. The pathogenicity of fowl-pest strains for ferrets paralleled their pathogenicity for mice. All four strains produced cross-immunity in ferrets. The clinical symptoms produced in ferrets by fowl-pest virus were those of ascending myelitis. At autopsy there were also broncho-pneumonia and lesions of the liver, kidney and stomach. No relation could be found between the virus of fowl-pest and other viruses—canine distemper, influenza, Rift Valley fever—which induce broncho-pneumonia in ferrets. The pig inoculated intranasally with fowl-pest virus exhibits no reaction but develops immune bodies; inoculated intracerebrally it develops a short febrile reaction.

CENTRIFUGATION STUDIES. III. The viruses of foot-and-mouth disease and vesicular stomatitis. W. J. Elford and I. A. Galloway. Brit. Jour. Exp. Path., xviii (1937), p. 155.

The inverted capillary tube technic for studying the behavior of suspensions in centrifugal fields has been successfully applied

in studies with the virus of foot-and-mouth disease, one of the smallest of known animal viruses. Measurements of the rate of sedimentation of the virus are recorded; its particle size is calculated to be 20 millimicrons. The particles are relatively uniform in size; all strains were found to have the same particle size. Measurements of the rate of sedimentation of the virus of vesicular stomatitis also were recorded. The particle diameter of this virus was found to be 74 or 60 millimicrons, according to whether the density of the particles was assumed to be 1.20 or 1.30, respectively.

CHEMICAL PROPERTIES OF THE PURIFIED SPREADING FACTOR FROM TESTICLE. Albert Claude and F. Duran-Reynals. Jour. Exp. Med., lxv (1937), p. 661.

The factor in testicular extracts responsible for enhancing infections and increasing tissue permeability was found to be soluble in water, in salt solution and in acid media. It is relatively stable at high pH concentrations and it is not precipitated or inactivated by hydrochloric acid up to a pH of 2.0. The substance is not soluble in acetone, ether, alcohol, chloroform or pyridine. It is inactivated by crystalline trypsin and pepsin at the optimum pH of action of these enzymes. It is not attacked by a crystallized carboxypolypeptidase. The substance does not pass semipermeable membranes which retain proteins. The color tests for proteins are positive. At least 14.2 per cent of the fraction isolated is nitrogen. Taken together these properties are indicative that the testicular factor is a protein. Concentrated solutions of the factor produce a condition of the skin having the characters of edema.

SPONTANEOUS ENCEPHALOMYELITIS OF MICE, A NEW VIRUS DISEASE. Max Theiler. Jour. Exp. Med., lxv (1937), p. 705.

After intracerebral injection of the virus of mouse encephalomyelitis and an incubation period varying from seven to over 30 days, a flaccid paralysis of one of the limbs appears. This paralysis spreads rapidly until all four limbs are affected. Young mice are more susceptible than old; very young mice often die without signs of paralysis. Intranasal instillation of the virus is the only other method of producing the infection. This method produces paralysis in only a small percentage of the mice. Paralysis in surviving mice recedes gradually but a permanent residual paralysis is almost invariable. Such mice are

virus carriers, as the virus can be recovered from the spinal cord for one year after infection. Recovery from infection results in immunity to subsequent inoculation. The virus resists the action of 50 per cent glycerin at from 2 to 4° C. for at least 150 days. It passes all grades of Berkefeld filters with ease. The particle size of the virus is probably about 13 to 17 millimicrons. The virus is not pathogenic for Rhesus monkeys. The anatomical basis of the paralysis is an acute necrosis of the ganglion cells of the anterior horn of the spinal cord and isolated ganglion cells of the cerebrum.

EFFECT OF PARATHYROID EXTRACT AND CALCIFEROL ON THE TISSUES OF THE NEPHRECTOMIZED RAT. F. A. McJunkin, W. R. Tweedy and E. W. McNamara. Amer. Jour. Path., xiii (1937), p. 325.

A comparison of parathyroid hormone and calciferol effects in bilaterally nephrectomized rats reveals that although both the hormone and the calciferol loosen calcium and remove it from the bone, it is only calciferol that effected a hypercalcemia during the 48-hour experimental period. In the bilaterally nephrectomized rat, the administration of large doses of parathyroid extract did not produce a characteristic rise in serum calcium but did cause a pronounced osteoclastic bone resorption within 48 hours. The subcutaneous injection under similar conditions of calciferol caused a hypercalcemia and also osteoclastic bone resorption within 48 hours.

THE BREEDING MEDIA OF SOME COMMON FLIES. M. Thomas and C. Hammer. Bul. Entom. Res., xxvii (1937), p. 559.

A great number of observations on farms in Denmark showed that, during the summer, *Musca domestica* (the common house fly) breeds in the first place in fresh pig dung deposited in manure pits, secondly in the bedding of calf-stables, and next in horse-boxes and pig-sties. Horse dung in manure pits is of inferior importance for house fly breeding under rural conditions in Denmark. *Stomoxys calcitrans* (the stable fly) breeds chiefly in the bedding of calf-boxes, and to a much lesser extent in humid remains of straw, old fodder or other vegetable material left in corners and crevices of stables. *Stomoxys* larvae were only rarely found in horse manure, pig dung or cow dung. Oviposition in the open was not observed and if it exists is quantitatively of no importance. Four experiments were made in which vessels

with pig dung, horse dung, calf dung and cow dung were exposed to the oviposition of flies. The number of flies subsequently hatching from each vessel gave an idea of the preference of each species. There was no essential difference between horse manure and pig dung in their power to attract the ovipositing flies; calf dung was less attractive and no eggs were laid in cow dung. *Stomoxys* preferred oviposition in horse manure and calf dung; only few eggs were laid in pig and cow dung.

TETANUS IMMUNITY: THE RESISTANCE OF GUINEA PIGS TO LETHAL SPORE DOSES INDUCED BY ACTIVE AND PASSIVE IMMUNIZATION. P. A. T. Sneath, E. G. Kerslake and F. Scruby. Amer. Jour. Hyg., xxv (1937), p. 464.

Computable numbers of tetanus spores have been shown to induce lethal tetanus in unprotected guinea pigs when mixed with a 50 per cent solution of calcium chloride and injected intramuscularly. This has been designated as a lethal spore dose. Guinea pigs within four to eight weeks after being injected with tetanus toxoid are more effectively protected from death and other signs of tetanus following a lethal spore dose of *Clostridium tetani* than animals similarly infected and passively protected by a single dose of tetanus antitoxin. In two groups, each of 55 animals of the actively immunized, 9 per cent died of tetanus and 80 per cent were completely protected; whereas of the passively protected, 38 per cent died and 24 per cent were completely protected. An antitoxin level of approximately 0.01 unit per cubic centimeter of serum would appear to be the critical level desirable of attainment in the active immunization of guinea pigs. Since infection with *Clostridium tetani* is a localized process, it is probable that such an antitoxin level as will prevent the manifestation of tetanus in guinea pigs would also be sufficient to prevent the disease in man.

AN INVESTIGATION OF THE INVASIVENESS OF A STRAIN OF STREPTOCOCCUS HEMOLYTICUS. M. G. Pradhan. Brit. Jour. Exp. Path., xviii (1937), p. 90.

A spreading factor is elaborated by the cocci in the presence of serum during the early period of growth. This substance is distinct from hemolysin; a 16-hour filtrate did not possess any spreading factor. As growth proceeds, this factor is destroyed or neutralized, possibly by the products of metabolism. It is elaborated only in media containing a high proportion of serum. The necessity of serum suggests that it can be produced even in

greater proportions in the animal body. The spreading factor has an enhancing action upon the power of an avirulent strain to produce an inflammatory lesion in rabbit's skin. The factor is probably in close association with the surface of the cocci. The author concludes that the invasive power of this streptococcus seems to be the capsular material evolved by the cocci only in the very early stages of multiplication; this substance may enhance the resistance of the microörganism to phagocytosis; free in the surrounding medium it favors the spread through the host's tissues.

Visitors at the Journal Office

An unusually large number of prominent veterinarians have visited the JOURNAL office during recent months. Among the number were: Dr. H. D. Bergman, Ames, Iowa, chairman of the A. V. M. A. Committee on Proprietary Pharmaceuticals; Dr. Oscar V. Brumley, Columbus, Ohio, president-elect of the A. V. M. A.; Dr. D. M. Campbell, Chicago, chairman of the A. V. M. A. Committee on Public Relations; Dr. L. Enos Day, Chicago, secretary of the United States Live Stock Sanitary Association; Col. Robert J. Foster, Washington, D. C., president of the A. V. M. A.; Dr. Robert Graham, University of Illinois, Urbana, Ill.; Dr. Wm. A. Hagan, Dean, New York State Veterinary College, Ithaca, N. Y.; Dr. C. M. Haring, University of California, Berkeley, Calif.; Dr. Reuben Hilty, Toledo, Ohio, former president of the A. V. M. A.; Dr. C. D. Lowe, U. S. Department of Agriculture, Washington, D. C.; Dr. N. S. Mayo, Highland Park, Ill., chairman of the A. V. M. A. Committee on Education; Dr. J. R. Mohler, Chief of the U. S. Bureau of Animal Industry, Washington, D. C.; Dr. Carl J. Norden, president of Norden Laboratories, Lincoln, Neb.; Dr. Carl Olson, Jr., New York State Veterinary College, Ithaca, N. Y.; Dr. E. A. Rodier, Pullman, Wash.; Dr. A. S. Schlingman, Parke, Davis and Co., Detroit, Mich.; Dr. W. T. Spencer, Omaha, Neb., chairman of Committee on Local Arrangements.

Meat Production Declines

Meat production for the first five months of 1937 decreased almost 1,000,000,000 pounds from the average of the five-year period 1928-1932, according to estimated figures. There was an increase in the production of beef, veal, lamb and mutton, but this was more than offset by the decrease in pork, which was off about 32 per cent from the five-year average.

Regular Army

Captain Russell McNellis is directed to proceed at the proper time from Washington, D. C., to Brooklyn, N. Y., and report to the commanding general, New York Port of Embarkation for temporary duty as transport veterinarian on the USAT Ludington for the trip leaving New York on or about May 20, 1937, for San Francisco, Calif., and upon arrival report in person to the commanding general, Ninth Corps Area, for temporary duty pending the sailing of the July 7, 1937, transport from San Francisco for New York, and sail on that transport; upon arrival in New York return to his proper station, Washington, D. C.

1st Lieut. Thomas C. Jones is directed to proceed at the proper time from Presidio of Monterey, Calif., to Fort Mason, Calif., reporting to the commanding general, San Francisco Port of Embarkation not later than July 27, 1937, for temporary duty as transport veterinarian on the USAT Ludington to accompany the shipment of animals at that time from San Francisco, Calif., to New York, and return from New York to San Francisco on the USAT Ludington, and upon arrival return to his proper station, Presidio of Monterey.

1st Lieut. Andrew J. Sirilo, Fort Mason, Calif., is designated as transport veterinarian on the USAT Meigs for the trip leaving San Francisco, Calif., for the Philippines June 24, 1937, and returning August 23, 1937, only.

1st Lieut. Lloyd C. Tekse, now under orders for duty at the San Francisco Port of Embarkation, is assigned as transport veterinarian on the USAT Ludington for the trip leaving San Francisco for New York on or about November 20, 1937, and will be continued on this duty until the shipment of animals to Panama is completed, at which time he will return to his proper station, San Francisco Port of Embarkation, Fort Mason, Calif.

Major Louis G. Weisman is relieved from further assignment and duty at Fort Clark, Texas, effective on or about July 5, 1937, and directed to proceed to Fort Ethan Allan, Vt., for duty.

Orders relieving 1st Lieut. Albert A. Roby, Jr., from duty at Fort Bliss, Texas, and assigning him to duty at the Army Veterinary School, Army Medical Center, Washington, D. C., is revoked.

1st. Lieut. Edwin L. Millenbruck is relieved from duty at Fort Bragg, N. C., effective in time to proceed to Washington, D. C., and report to the commanding general, Army Medical Center, on or about August 28, 1937, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School.

Lt. Col. George W. Brower is relieved from assignment and duty at Fort Francis E. Warren, Wyo., effective on or about August 1, 1937, and directed to proceed to Fort Clark, Texas, for duty.

Major John K. McConeghy is relieved from his present assignment and duty at Fort Bliss, Texas, on or about June 27, 1937, and will then proceed to his home and await retirement. Major McConeghy will be retired for disability.

The promotion of Major Chauncey E. Cook to the grade of lieutenant colonel, with rank from May 24, 1937, is announced.

Veterinary Reserve Corps

NEW ACCEPTANCES

Holm, Glenn C.....1st Lt...Box 25, Rexburg, Idaho.
Thome, Andrew L.....1st Lt...488 Edgewood Ave., N. E., Atlanta, Ga.

SEPARATIONS

Washburn, Robert E.....Capt...Gloversville, N. Y. Died April 13, 1937.

PROMOTIONS

To

Hodgson, Melvin E.....Capt...2912 N. W. 18th St., Oklahoma City, Okla.
Starnes, Mervyn B.....Capt...Care City Hall Annex, Dallas, Texas.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Cavanaugh, Joseph Leo.....1st Lt...Omaha, Neb.
Schmidt, Martin Geo.....1st Lt...Fort Leavenworth, Kan.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Fly, Glenn O.....1st Lt...Fort Knox, Ky.
Michael, Lloyd J.....1st Lt...Fort Leavenworth, Kan.
McDonald, Alvin R.....1st Lt...Fort Adams, R. I.

Examination for Veterinary Meat Inspector

The California State Personnel Board announces an examination for a

VETERINARY MEAT INSPECTOR

to obtain eligibles to certify to several positions now filled by temporary employés; and to establish an eligible list from which to fill future vacancies.

The age limits are 21 to 50 years. Graduates of recognized veterinary colleges are eligible. Experience in meat inspection or live stock disease control work is an additional desirable qualification. All applicants must be full United States citizens.

Examinations will be held at San Francisco, Los Angeles, San Diego and Sacramento on July 24, 1937. Applications must be made out on official application blanks, procurable in Sacramento at 404 Library-Courts Building; in San Francisco at 108 State Building; in Los Angeles at 401 State Building; at the San Diego Civil Service Commission, Library Annex, 9th and E Streets, or by writing to any of these offices.

Veterinarians and the Social Security Act

Veterinarians employing one or more persons have been advised by Commissioner of Internal Revenue Guy T. Helvering to make immediate tax returns as required under the provisions of Titles VIII and IX of the Social Security Act to avoid further payment of drastic penalties which are now accruing.

Commissioner Helvering has pointed out that every person employed by a veterinarian comes under the provisions of Title VIII, which imposes an income tax on the wages of every taxable individual and an excise tax on the pay-roll of every employer of one or more. This tax is payable monthly at the office of the Collector of Internal Revenue. The present rate for employer and employé alike is 1 per cent of the taxable wages paid and received.

Under Title IX of the Act, employers of eight or more persons must pay an excise tax on their annual pay-roll. This tax went into effect on January 1, 1936, and tax payments were due from the employers, and the employers alone, at the office of the Collector of Internal Revenue on the first day of January, 1937. This tax is payable annually, although the employer may elect to pay it in regular quarterly installments.

The employer is held responsible for the collection of his employé's tax under Title VIII, and is required to collect it when the wages are paid the employé, whether it be weekly or semi-monthly. Once the employer makes the 1 per cent deduction from the employé's pay, he becomes the custodian of federal funds and must account for them to the Bureau of Internal Revenue.

This is done, Commissioner Helvering explained, when the employer makes out Treasury form SS-1, which, accompanied by the employé-employer tax, is filed during the month directly following the month in which the taxes were collected. All tax payments must be made at the office of the Collector of Internal Revenue in the district in which the employer's place of business is located.

Penalties for delinquencies are levied against the employer, not the employé, and range from 5 per cent to 25 per cent of the tax due, depending on the period of delinquency. Criminal action will be taken against those who wilfully refuse to pay these taxes.

Employers of one or more persons are also required to file Treasury forms SS-2 and SS-2a. Both are informational forms and must be filed at Collectors' offices not later than July 31, 1937, covering the first six months of the year. After that, they are to be filed at regular quarterly intervals. Form SS-2 will show all of the taxable wages paid to all employés, and form SS-2a the taxable wages paid each employé.

Associated Serum Producers Extend Educational Campaign

Plans for the continuation of the educational and advertising campaign of the Associated Serum Producers, Inc., for another year and a half were announced the past month by the Executive Committee of the organization, following a formal ballot taken among all member companies. This move follows widespread approval of the current campaign, by both practicing veterinarians and veterinary organizations throughout the country. The new campaign will follow the same lines as the one now in progress, except that it will be on a larger scale, and will continue until January 1, 1939.

According to the new budget recently approved by member companies, the 1937-38 effort will include news releases on animal health subjects to over 1,000 newspapers and farm publications; the furnishing of a weekly feature column, "Health Hints for Live Stock," to 800 newspapers which have requested this feature; two series of informative talks over leading radio stations —one in summer and the other in winter; a spring campaign of spot announcements on radio stations about pig-weaning time; a series of paid advertisements in eleven farm journals in the spring and fall, and further distribution of the booklet on "Live Stock Health" through practicing veterinarians.

Most of this work will be carried on through the American Foundation for Animal Health, which has been underwritten by the Associated Serum Producers, Inc., to disseminate such educational material. The entire campaign will be designed to acquaint owners of live stock with the dangers of various animal diseases, and to urge them to consult veterinarians for diagnosis and treatment.

At the request of a number of state veterinary associations, copies of the news releases and other educational material will also be furnished to many state associations for distribution to the press. This is expected to extend further the scope and influence of the campaign. Plans have ben made for an extensive exhibit of the Associated Serum Producers, Inc., educational program at the Omaha convention next month, so that veterinarians throughout the country may see the work that is being done in their behalf.

Trichina Educational Program

Dr. J. R. Mohler, chief of the U. S. Bureau of Animal Industry, has directed each inspector-in-charge to designate one member of his force, preferably an inspector engaged in hog cholera control, to contact farmers and other swine-growers for the purpose of outlining methods of controlling trichinosis in hogs. Dr. Mohler very properly directs attention to the fact that the better control of trichinosis in hogs will have financial advantages for the producers of hogs and at the same time provide a measure of greater safety for human health.

The U. S. Bureau of Animal Industry has issued leaflets, posters and press releases in efforts to inform the public regarding the danger of trichinosis, which may follow the consumption of draw or improperly cooked pork. Repeated outbreaks of trichinosis in man can have nothing but an unfavorable effect on the consumption of pork and this will react to the disadvantage of the producer.

Sources of trichinosis in swine include the following:

(1) The feeding of raw garbage to swine.

(2) The feeding of offal from slaughter-houses.

(3) The feeding of contents of scrap-barrels.

(4) The failure to bury, burn, or otherwise properly dispose of hogs, dogs, cats, rats and other animals which die in lots and pastures, and which may be eaten by hogs.

If swine-growers will follow the principles of the Swine Sanitation System formulated by the Bureau, Dr. Mohler points out, trichinosis may be eliminated entirely, as most of the known sources of trichinæ are precluded by the system. At the same time, the cost of producing pork may be lowered through better gains in growth and earlier finishing for market.

COMMENCEMENTS

UNIVERSITE DE MONTREAL

Six graduates from l'École de Médecine Vétérinaire de la Province of Québec, Université de Montréal, received the degree of Doctor of Veterinary Medicine, at the 1937 commencement exercises. They are:

Mozart Clairmont
Edmond Doyon
Gérard Laberge
Bernard Lasalle
Roger Privée
Henri Troalen

Edmond Doyon and Henri Troalen were graduated with great distinction, and Gérard Laberge and Bernard Lasalle with distinction.

ALABAMA POLYTECHNIC INSTITUTE

The sixty-fifth annual commencement exercises of the Alabama Polytechnic Institute were held at Auburn, May 31, 1937. The commencement address was delivered by John Temple Graves, II, Litt.B., LL.B., D.C.L., columnist, *Birmingham Age-Herald*, Birmingham, Ala. In the School of Veterinary Medicine, 25 candidates received the degree, Doctor of Veterinary Medicine:

Hubert Leighton Allen
Jap Calvin Allen
Norman George Baird
Conway C. Brock
George Cohen
William Lawrence Cox
Louis Fink
Silvio Anthony Fittipaldi
Woodrow Wilson Harkins
Solomon Nathan Havel
Howard Ellery Hill
Elliot Kaplus
Martin George Lorber
James McLain McKinnon
Ellis James Martin
Clyde Robert Moses
Wendell Lamar Pinckard
Edmund R. Price
Charles Speer Roberts
Irving Jay Sadow
Daniel Briggs Schlosser
Jay Neff Thomas, Jr.
Cecil Paul Vickers
Edward Douglas Walters
Theron Williams

KANSAS STATE COLLEGE

The seventy-fourth annual commencement exercises of Kansas State College were held at Manhattan, May 31, 1937. Hon. Walter A. Huxman, Governor of Kansas, delivered the commencement address. In the Division of Veterinary Medicine, 29 candidates received the degree, Doctor of Veterinary Medicine. They are:

*Aaron Francis Allison
*Guy William Bayles
*Clark Wayne Burch
*Lucius Nelson Butler
Horace Reynolds Collins, Jr.
Edwin Morris Crawford
Walter Wallace Fechner
*Pearl Hugh Hand
*Henry Everett Harriman
*Earl Hester Harrison
*John Graham Hemphill
*William Hugh Hervey
Harry Ethelburt Hubbard
*Irvin Preston Irwin
*James Randle Ketchersid
*George Kendrick Lang
*Russell Martin Madison
Floyd Edward Monroe
*Richard Eugene Omohundro
Ray Sherman Pyles
*Lee Thomas Railsback
Ward Dallas Redman
*Charles Edwin Robinson
Lawrence Eric Spong
Clifford Wesley Turner
*Irwin John Twiehaus
*Willard Merrill Van Sant
James Howard Watson
*Ben N. Winchester

Nineteen members of the graduating class (indicated by *) received commissions in the Veterinary Officers Reserve Corps of the United States Army.

High honors were awarded to Edwin Morris Crawford. Honorary recognition was also given to Guy William Bayles and Floyd Edward Monroe.

UNIVERSITY OF PENNSYLVANIA

The 181st annual commencement exercises of the University of Pennsylvania were held on June 9, 1937. Forty-seven students were awarded the degree of Doctor of Veterinary Medicine as follows:

Walter Millard Andress
William Powell Barchfeld
*Arthur Valentine Bartenslager
Harry Francis Bicking Bartolett
Irving Hershel Botwinick
James Robert Brown
*John Patrick Cashin
John Etherington Craige
David William Crisman
Blakey Thatcher Deal
Joseph Dictor
James Garfield Eagelman, Jr.
Russell Stanley Edmonds
Homer Smith Forney
John Carroll Fowble
John Raymond Francesila
James Auten Frazee
Philip Henry Gerlach
*William Sylva Gochenour
Bernard Goldis
Arthur Nathaniel Gorelick
Melvyn Dexter Hale
George Richard Harms
Wilson Roberts Haubrich
*Harry Morris Hershman
Walter Brockett Holcomb
Hugh Huff Keeler
Vaughan Collins Lancaster
Harold Conrad Lower
Louis John Maher
Julio John Malnati
Leo Richard McKinney
William Marvel Moore
*Jesse Bartolet Moyer
Edward Charles Preston
Melvin Moses Rabstein
*Lester Reddin, Jr.
Carl Morris Richter
Raymond Richard Rohrer
Sydney Rosenberg
Israel M. Saturen
William Jacob Schuck, Jr.
Robert John Stadler
Benjamin Franklin Stearn
Donald Winfred Stever
Douglas Fleming Watson
Wayne Clinton Weaver

Saul Pravitz received his degree in February, 1937.

*With honors.

The J. B. Lippincott Prize of $100, for the highest general average in examinations during the four years, was awarded to William S. Gochenour and Harry M. Hershman.

The Jeanette Blair Prize of $50, for the best work done in the Small-Animal Clinic, was awarded to Lester Reddin, Jr.

The Leonard Pearson Prize of $50, to the member of the senior class, who, in the judgment of the faculty of the School of Veterinary Medicine, was best qualified to receive it, was won by Arthur V. Bartenslager.

The 1930 Class Prize of a book, to the member of the senior class for the highest average in the courses in surgery, went to John P. Cashin and Jesse B. Moyer.

The Suburban Cattle Breeders' Association Prize of $25, to the member of the sophomore class who attained the highest average in the course of physiology, was awarded to Russell C. Jones, Jr.

The Anatomy Prize of the Class of 1926, to the member of the sophomore class for the best work done in the courses in anatomy, was awarded to Ferner Sellers.

The Twelfth International Veterinary Congress Prize of a copy of the Proceedings of the Twelfth International Veterinary Congress, to the member of the senior class who, in the judgment of the faculty, was the outstanding student of the class, to Arthur V. Bartenslager.

Martin A. Kaplan was awarded the 1914 Class Prize for attaining the highest general average for the freshman year.

IOWA STATE COLLEGE

The annual commencement exercises of the Iowa State College were held June 14, 1937. The degree, Doctor of Veterinary Medicine, was conferred upon the following students:

Everett Theodore Anderson
Floyd William Bertels
Leonard Allen Bowstead
Raymond Nicholas Brenny
William Everett Brown
James Albert Butterworth
Don Leory Caswell
Donald Rolfe Cordy
Cornelius Andrew DeValois
Lewis Albert Dykstra
Robert Frank Freeman
Lloyd Vern Fry
Paul Cecil Garner
Raymond Albert Gathmann
Ollie Revels Grantham
Charles Gaylord Hanna
Ray Davenport Hatch
Isaac Edward Hayes
Emil Laurence Koch
August William Krause
Paul Ernest Maland
Lawrence Minsky
William Seastream Monlux
Russell Loy Oettiker
Howard Charles Raven
Benjamin Rosenfeld
Carl John Schubert
Piao Sheo
Benjamin Joseph C. Sinkler
Herman Harold Soehl
Warren Bixby Thurber
Don Carrol Vest
George Miner Wilson
*Stanley Nixon Wood

*Received degree, June 22, 1936.

Dr. Carlos Rosenbusch (Iowa '35) received a Master of Science degree in veterinary bacteriology.

Stanley Nixon Wood was the honor student in veterinary medicine, as well as holding the highest all-college average, thereby winning the George Judisch Award. This award consists of the initiation fee and membership dues in the A. V. M. A. for five years.

The winners of the G. G. Graham Prizes, awarded to outstanding students in clinical medicine, were William E. Brown, first, and Benjamin J. Sinkler, second.

COLORADO STATE COLLEGE

The fifty-fifth annual commencement exercises of Colorado State College were held June 1, 1937. Dr. Thurston Davies, President of Colorado State College, delivered the commencement address. The following candidates were awarded the degree, Doctor of Veterinary Medicine:

Frank X. Gassner
Philip McClave
Jim C. McIntyre
Otto Montgomery
Linn Poley
Fred Noah
Jordan Rasmussen

Scholastic honors were awarded to Frank Gassner.

MICHIGAN STATE COLLEGE

The seventy-ninth annual commencement exercises of the Michigan State College were held June 14, 1937, and the following students received the degree, Doctor of Veterinary Medicine:

John J. Arnold
Theodore M. Barr
Erwin A. Bortman
James L. Boydston
Breyton D. Brady
Charles H. Failing
Luther E. Fredrickson
Milo J. Henshaw
Russell J. Henshaw
Hans H. Ruhland
Lyle F. Spake
Edmund E. Visger
Josephine R. Von Eberstein
Ernest S. Weisner
Alvin R. Wingerter

John J. Arnold was graduated with high honors.

The Michigan State Veterinary Medical Association prize of $25, to the senior who has done the best work during his course, was awarded to Alvin R. Wingerter. The set of Proceedings of the Twelfth International Veterinary Congress was received by John J. Arnold.

Ralph H. Benedict and Donald F. Freshour were graduated at the close of the summer session of 1936.

OHIO STATE UNIVERSITY

The sixtieth annual commencement exercises of the Ohio State University were held on June 14, 1937. The commencement address was delivered by Dr. George W. Rightmire, president of the University. The College of Veterinary Medicine presented 19 candidates for the degree, Doctor of Veterinary Medicine, as follows:

Everett Horton Akins
William Haye Armstrong
Howard Powers Buckley
David Harvey Carter
LeRoy Tenny Fisher
Albert Hathan Glassman
Garner Romaine Greenhoff
John Harry Helwig
Wilfred Clinton Jackson
Wesley Orville Keefer
Ernestine Gill Kelly
Willis Edward Lord
Edward Thomas Marsh
Carl Max Oldham
Louis Joseph Payen
Harold Oscar Sheet
David Splaver
John W. Vandeven
Thomas Joseph Wheelin

Dr. Robert P. Wagers (O. S. U. '36) received a Master of Science degree in veterinary pathology.

President Rightmire conferred the degrees, following the formal presentation of the candidates. The diplomas were awarded by Dean Oscar V. Brumley.

STATE COLLEGE OF WASHINGTON

The annual commencement exercises of the State College of Washington were held June 14, 1937, with an attendance of 2,000 people. The commencement address was delivered by the governor of the state, Hon. Clarence D. Martin. The following students from the College of Veterinary Medicine were awarded their degrees:

Franklin F. Christofferson
Ernest Donald Copple
John Glynn Cranfield
Stuart MacKenzie Dingwall
Kenneth Erickson
Virgil Holden Fisher
George Chase Folger
Clinton Leslie Gould
Donald Dean House
Marion Lane Ledgerwood
Orville Beverly McRory
Hilding Mauritz Marlowe
William Ernest Mottram
Lyle G. Nicholson
Wilson Munn Pang
Emmet William Paul
Werner Pundt
Robert Samuel Russ
Albert Andrew Taylor
Lewis Kay Todd
Edward James Watson

Lyle Nicholson and Edward Watson received their degrees with highest honors, and Donald House, John Cranfield and Virgil Fisher received theirs with honors.

Birdsall Carle, a junior veterinary student, was awarded the Alumni Scholarship for the year 1937-38.

CORNELL UNIVERSITY

The sixty-ninth annual commencement exercises of Cornell University were held June 21, 1937. Dr. Livingston Farrand, president of the University, delivered the commencement address. The New York State Veterinary College presented 34 candidates for the degree, Doctor of Veterinary Medicine, as follows:

Albert Louis All
Jeroham Asedo
Jack Edward Baker
Francis Arthur Barry
James Alan Brennan
Dorsey William Bruner
Albert Johnson Crane
Salvatore Matthew Dragotta
Arnold Eder
Elwood Stephen Francis
Irving Grodin
Thomas Edmond Hickey
Oby Jared Hoag
Seymour Hoffman
Donald Vincent Hughes
Norman Henry Johnson
Carl Kellman
Marie Agnita Koenig
Leon Jules Lacroix
Murray Jack Lavitan
Gerard Paul Lemire
Abraham Alan Levinson
Harry Aaron Lutvack
Alfred Francis Nolan
George Greenwood Pickett
Herbert Racoff
Irwin Herbert Roberts
Jay Jewell Sarasohn
George Conrad Schloemer
Max Martin Shindel
Morris Siegel
Sam Stieber
Herman Tax
Victor Frost VanWagenen

Gordon Darrow Boyink received his degree in February, 1937.

A. AND M. COLLEGE OF TEXAS

The sixty-first annual commencement exercises of the Agricultural and Mechanical College of Texas were held June 4, 1937. The following students in the School of Veterinary Medicine were awarded their degrees:

Joseph Marion Black, Jr.
James Nelson Brown
Frank Wilfred Brundrett
Daniel Cecil Frederick
Edgar Alfred Grist
Thomas Hudson Hawkins
Wiley Henry Horn
R. S. Martin
Homer T. Martinez
John Freeman Melton
Francis J. Schott
Charles Roy Wainright
Harvey S. Widney
Horace Randolph Willard
Isaiah Geoffrey Wirtz, Jr.

Eleven of the class plan to enter practice, three have accepted positions with the Texas Live Stock Sanitary Commission and one will go into commercial work.

For he that once is good, is ever great.—Ben Jonson.

COLORADO STATE COLLEGE VETERINARY SHORT COURSE

The most successful short course in the history of the institution was held at the Colorado State College, Fort Collins, May 19-21, 1937. Not only was the attendance much larger than at any previous session, but the completeness and diversity of the program was such as to hold the keenest interest of those in attendance throughout three full days.

Following addresses of welcome by Dean I. E. Newsom and President Chas. A. Lory, a splendid résumé of his experience with equine encephalomyelitis and allied conditions was presented by Dr. George H. Carr, of Brighton. It was pointed out that, in Colorado, outbreaks of this malady have been seasonal and restricted primarily to river valleys and irrigated areas, which seems to support the contention that mosquitoes are the principal vectors.

Nervous diseases of lower animals were systematically discussed in a well-illustrated talk by Dr. Carl F. Schlotthauer, of the Mayo Foundation, Rochester, Minn. Correlation of nervous structure and function was splendidly analyzed. In later lectures, Dr. Schlotthauer covered the diseases of the eye and digestive disorders of the dog. During the clinical program he contributed valuable discussions and operations.

Dr. T. H. Ferguson, of Lake Geneva, Wis., in a series of lectures and clinical demonstrations, covered many of the diseases and surgical procedures incident to bovine practice.

Other visiting veterinarians who gave very helpful papers or lectures were Dr. Frank Breed, of Lincoln, Neb., who covered, in his usually effective manner, nutritional diseases of swine, and hog cholera and allied infections of hogs; Dr. W. H. Hendricks, Salt Lake City, Utah, who, as State Veterinarian of Utah, gave an exhaustive analysis of his experience in Bang's disease control, and Capt. C. D. Greer, Fort Robinson, Neb., who, as veterinary officer in charge of the Army breeding station, discussed breed-

ing practices and diseases of foals. Dr. Hendricks' paper was discussed by Dr. H. D. Port and Dr. R. M. Gow, state veterinarians of Wyoming and Colorado, respectively, as well as by Drs. W. A. Sullivan and A. H. Francis, federal veterinarians in charge of Wyoming and Colorado, respectively.

Dr. H. E. Kingman, of the Wyoming Hereford Ranch, Cheyenne, Wyo., gave interesting talks and demonstrations bearing upon semen studies, artificial insemination and breeding problems in cattle.

An interesting demonstration of x-ray and fluoroscopic equipment, such as is now in use at the College Hospital, was presented by Mr. R. V. Wood, of Denver.

Other papers were presented by Drs. I. E. Newsom and R. F. Bourne, of the College faculty, and clinical demonstrations were given by numerous veterinarians, including the visitors from other states, as well as by Drs. Geo. H. Oliver, N. J. Miller, A. N. Carroll, H. G. Weigand and others from Colorado. Dr. James Farquharson, head of the Department of Surgery, should be given highest praise, not only for arranging a splendid program, but for the highly satisfactory facilities provided for carrying it to completion.

Social diversions were provided in the form of an informal dinner, a dinner dance and a ladies' trip to Rocky Mountain National Park, for arranging which, representatives of several of the leading veterinary supply-houses deserve much credit.

A short business meeting of the Colorado Veterinary Medical Association resulted in the adoption of a new constitution and by-laws and the reëlection of present officers to carry on until the conclusion of the present fiscal year. Registration of veterinarians in attendance totaled 103.

R. F. BOURNE, *Reporter*.

VETERINARY ALUMNI SOCIETY, UNIVERSITY OF PENNSYLVANIA

The Alumni Society of the School of Veterinary Medicine, University of Pennsylvania, celebrated the fiftieth anniversary of its founding June 4-5, 1937, at Philadelphia. An attendance of between 300 and 400 members and friends participated in the various activities planned for the group. The celebration was inaugurated Friday afternoon, with a split between the men and their wives, the ladies enjoying a visit to the Franklin Institute and a display presented in the planetarium, while their husbands

attended demonstrations at the Veterinary School. The demonstrations and presentations at the first session were as follows:

"Strongylosis of the Horse," by A. C. Wood.
"Autopsy Technic," by H. B. Bartolett.
"Fowl-Pox," by J. G. Eagelman.
"Autonomic Nervous System," by L. B. Fake, F. Sellers and W. H. Wright.
"Morphine Action," by P. L. Gaunt, Jr., and I. G. Cashell.
"Barium Chloride in the Horse," by R. G. Buckingham and J. G. Woodcock.

A high spot in the program occurred Friday evening in the form of a dinner-dance held at the Bellevue-Stratford Hotel. With the president of the Society, Dr. H. W. Barnard, acting as toastmaster, the group was favored with a most satisfying dinner and an entertaining floor show. The principal speaker of the evening, Professor Cornelius Weygandt, of the English department, succeeded in adding a fitting charm to the occasion with his artistic description of "The Country Heart."

Saturday, general Alumni Day at the University, brought clinical demonstrations in the morning with W. P. Barchfield, H. H. Keeler and D. W. Stever, on the large-animal detail, and L. Reddin, Jr., L. R. McKinney and J. A. Frazee with the small animals. This meeting was under the supervision of Dr. H. P. Eves, class of '87.

A business meeting followed the clinics and preceded the luncheon, always an occasion for numerous activities. At this time prizes were awarded by the Society to the member who had traveled the greatest distance to attend, Dr. W. C. Prouse, of Minneapolis, Minn., and to the "runner-up," Dr. F. F. Buck, of Rural Retreat, Va. A prize went to the class of '87 for the largest proportion (67 per cent) of members present.

The annual presentation of student prizes also was made at this time by Dean George A. Dick. The luncheon was followed by the Penn-Princeton baseball game at Franklin Field.

A. H. CRAIGE, *Reporter.*

MICHIGAN STATE VETERINARY MEDICAL ASSOCIATION

The 55th annual meeting of the Michigan State Veterinary Medical Association was held at the Michigan State College, East Lansing, June 22-23, 1937. Dr. J. William Hansen, of Greenville, presided at all sessions. The literary program was as follows:

"Mastitis," by Dr. Ashe Lockhart, Kansas City, Mo.
"Developments in Equine Practice," by Dr. D. M. Campbell, Chicago, Ill.
"Canine Nutrition," by Dr. H. E. Robinson, Chicago, Ill.
"My Experiences in Cat Practice," by Dr. S. R. Elko, Highland Park, Mich.

Dr. J. P. Hutton, of Michigan State College, was in charge of the clinic. His assistants were Drs. A. E. Erickson, Charlotte; H. H. Clark, Lansing; A. V. Jandernoa, Pewamo; S. G. Colby, Monroe; H. Dykema, Muskegon; E. K. Sales, East Lansing, and F. D. Egan, Detroit. The clinic was conceded to be the most successful one which has been held at an annual meeting for several years.

The operations on large animals included three roaring operations, performed in the standing position; castration of a colt in the standing position; teat surgery on hard-milking cows, and demonstrating the use of the stomach tube in treating horses for worms.

Dr. J. A. Campbell, of Toronto, Ont., was the guest surgeon at the small-animal clinic. He demonstrated the Cesarean operation on bitches, the removal of tartar from dogs' teeth with a specially designed instrument, irrigation of the anal pouches and, in addition, gave several valuable pointers in small-animal practice. A conchal chondrotomy was demonstrated by Dr. H. Dykema, of Muskegon. Dr. E. K. Sales was in charge of x-ray demonstrations. Mr. C. M. Olson, of Chicago, conducted a dog-food demonstration which was especially interesting.

At the business meeting, a resolution was adopted protesting against the importation of meats from foreign countries where foot-and-mouth disease exists.

A vote on the next meeting place of the A. V. M. A. was decidedly in favor of Madison, Wis. A resolution was adopted discouraging the formation of veterinary coöperative organizations in counties adequately supplied with veterinarians. The Special Committee on Socialistic Medicine is to act in an advisory capacity concerning the organization of future veterinary coöperative organizations. Favorable action was taken toward the establishment of a Committee on Public Relations, which will coöperate with a like committee of the A. V. M. A.

The officers of the Ladies' Auxiliary provided entertainment and luncheons for visitors and members during the two-day session. A banquet was held for members, their wives, and guests on the evening of the first day, with an attendance of 135. Appropriate entertainment was provided.

Officers elected for the ensuing year were as follows: President, Dr. A. Z. Nichols, Pittsford; first vice-president, Dr. D. A. Curtis, Breckenridge; second vice-president, Dr. F. M. McConnell, Litchfield; third vice-president, Dr. H. Dykema, Muskegon; secretary-treasurer, Dr. H. H. Clark, Lansing, and member of Board of Directors, Dr. W. N. Armstrong (reëlected), Concord.

E. C. W. SCHUBEL, *Secretary-Treasurer.*

Capital Veterinarians Organize

Veterinarians located in the nation's capital have organized the District of Columbia Veterinary Medical Association, adopted a constitution and by-laws, elected officers and affiliated with the A. V. M. A. The officers for the current year are: President, Dr. Mason Weadon; first vice-president, Dr. H. W. Schoening; second vice-president, Maj. G. L. Caldwell, and secretary-treasurer, Dr. E. Barnwell Smith, all of Washington. Dr. Schoening has been chosen as delegate, and Dr. J. R. Mohler as alternate, to the A. V. M. A. House of Representatives.

Censure is the tax a man pays to the public for being eminent. —Swift.

THE CONVENTION PROGRAM

(*Continued from page* 11)

a roaring operation, a crytorchid operation and a trephine operation for the removal of polypus of the nasal cavity. Dr. E. A. Hewitt, of Iowa State College, will give a very interesting demonstration on the heart.

In the Swine Section, nutritional disorders will be given particular attention along with several of the more common diseases of swine. In the Ovine Section, there will be a number of practical demonstrations, including restraint, docking, castration, administering treatment for worms, and several others.

The Poultry Section, under the direction of Dr. C. D. Lee, of Iowa State College, has planned an elaborate program, featuring pullorum disease, tuberculosis, parasites, nutritional diseases, fowl-pox, laryngotracheitis, bronchitis, fowl cholera, fowl typhoid, leukosis, coccidiosis, and the grading of poultry.

In the Canine and Feline Section, there will be considerable of interest for veterinarians engaged in small-animal practice.

NECROLOGY

ALBERT C. GALBRAITH

Dr. Albert C. Galbraith, of Newville, Pa., died suddenly on February 22, 1937, of a heart attack. He had been a patient at Johns Hopkins Hospital, Baltimore, just before his death, which came while he was on a professional call.

Born at Mooredale, Pa., in 1880, Dr. Galbraith was a graduate of the Newville High School and the Kansas City Veterinary College, class of 1915. He practiced in Newville about twelve years and then entered the service of the Pennsylvania Bureau of Animal Industry. He returned to private practice early in 1936.

Dr. Galbraith joined the A. V. M. A. in 1927. He was a member of the Pennsylvania State Veterinary Medical Association and the Cumberland Valley Veterinary Club. He is survived by his widow (née Bertha Detweiler) and one daughter.

JAMES N. HORN

Dr. James N. Horn, of Peoria, Ill., died suddenly the night of May 28, 1937, apparently of a heart attack. He died before a physician could reach him.

Born at Cowden, Ill., November 25, 1890, Dr. Horn received his professional training at the Chicago Veterinary College. He was graduated in 1915 and practiced at Arrowsmith, Ill., before locating in Peoria.

Surviving Dr. Horn are his widow (née Ruth Swibold), his mother, one daughter, one sister and five brothers.

JAMES F. BARNES

Dr. James F. Barnes, of Toledo, Ohio, died at his home on May 10, 1937, after an illness of several years. He was 75 years of age.

Born in Wexford, Ireland, Dr. Barnes came to this country when he was only five years old. He was a graduate of the Department of Veterinary Medicine and Surgery of the Detroit College of Medicine, class of 1897, and had practiced in Toledo for about 39 years.

Dr. Barnes joined the A. V. M. A. in 1912. He was a member of the Twelfth International Veterinary Congress and Barton Smith Lodge, F. & A. M. His widow survives him.

EDWARD W. ROBERTS

Dr. Edward W. Roberts, of Lake Crystal, Minn., died at Sleepy Eye, Minn., on June 6, 1937, while attending a meeting. The cause of death was heart failure.

Born in Anglesea, North Wales, January 1, 1867, Dr. Roberts immigrated to Waukesha, Wis., as a young man of 22. Three years later, in 1892, he moved to Lake Crystal, Minn. After farming for more than 15 years, he decided to study veterinary medicine and entered the McKillip Veterinary College. Following his graduation in 1914, he returned to Minnesota and engaged in general practice at Lake Crystal.

Dr. Roberts is survived by his widow (née Sarah Ann Jones), three sons and two daughters. L. E. S.

GEORGE T. CROWLEY

Dr. George T. Crowley, of New Britain, Conn., was found dead in his damaged automobile, in Marlboro, the morning of June 7, 1937. It is believed that he suffered a heart attack as he was driving toward New London.

Born in New Britain, July 29, 1870, Dr. Crowley attended the Ontario Veterinary College. Following his graduation in 1901, he entered practice at New Britain. From 1905 to 1914, he was Meat and Food Inspector of his city. In 1917, he accepted a position with the State Department of Domestic Animals, a position he held at the time of his death. He did special work for the department in connection with rabies and other diseases.

Dr. Crowley had been a member of the Connecticut Veterinary Medical Association since 1901. He served as a member of the Board of Censors for a number of years. He was a member of Harmony Lodge F. & A. M. and of the Lodge of Elks of New Britain. He is survived by his widow, one daughter and two sisters. G. E. C.

WILLIAM JOHN ARMOUR

Dr. William J. Armour, of Goshen, Ind., died at his home on June 13, 1937, following an acute illness of only a few days. Death was due to angina pectoris from which he had been a sufferer for a number of years.

Born near Toronto, Canada, January 4, 1866, Dr. Armour was a graduate of the Ontario Veterinary College, class of 1890. He practiced first at Bangor, Mich., going to Goshen, Ind., about 40 years ago. He served as a member of the Common Council of Goshen from 1910 to 1914.

Dr. Armour joined the A. V. M. A. in 1901. He was a member of the Indiana Veterinary Medical Association and had served as president of that organization. He was a member of Goshen Lodge No. 12 F. & A. M.; Goshen Chapter 45, R. A. M.; Bashor Council 15 R. & S. M.; Goshen Commandery No. 50, Knights Templar; South Bend Consistory Scottish Rites, and the Shrine. He was also a member of the Goshen Lodge of Elks. He is survived by his widow (née Kathryn Bostetter), one daughter and one brother.

EARL D. ENSIGN

Dr. Earl D. Ensign, of Columbus, Ohio, dropped dead in Chillicothe, Ohio, on June 14, 1937. He had gone to Chillicothe to investigate some trouble among cattle on a farm near there, when he was stricken with a heart attack. He was 50 years of age.

Born in Defiance County, Ohio, Dr. Ensign studied veterinary medicine at Ohio State University. He was graduated in 1912 and practiced at Bryan, Ohio, for about 13 years, when he went to Toledo, to accept a position with the Board of Health there. He remained in this work until about 1928, when he located in Sylvania, Ohio. Four years ago, he accepted a position as field veterinarian with the Ohio Division of Animal Industry, at Columbus.

Dr. Ensign was a member of the Ohio State Veterinary Medical Association, Alpha Psi Fraternity and the Masonic lodge at Bryan, Ohio. He is survived by his widow, one son, one daughter, his parents, two brothers and one sister.

Our sympathy goes out to Dr. and Mrs. R. H. Tesdell, of Ogden, Iowa, in the death of their three-year-old son, Roger Leon Tesdell, in the Iowa Lutheran Hospital, Des Moines, on May 13, 1937, of spinal meningitis, after having been sick for six weeks.

PERSONALS

BIRTH

To Dr. and Mrs. Charles J. Griffin, of Springfield, Ohio, a son, Charles Thomas, May 8, 1937.

PERSONALS

Dr. Daniel Schlosser (Ala. '37) is practicing at Bourbon, Ind.

Dr. A. R. Bruner (Ind. '17) has removed from Oakland City, Ind., to Boonville, Ind.

Dr. Benner R. Criley (Iowa '28), of Ottumwa, Iowa, is now located at Pearl River, N. Y.

Dr. Len C. Henderson (K. C. V. C. '04) has removed from Spokane, Wash., to Temple City, Calif.

Dr. J. A. Winkler (Cin. '18), of Newport, Ky., is veterinarian to the Cincinnati Cat Club, recently reorganized.

Dr. C. P. Fitch (Corn. '11) has been reappointed a member of the Minnesota State Live Stock Sanitary Board.

Dr. John Ayer (Cin. '18) gives a change of address from Cincinnati, Ohio, to 221 Dayton Avenue, Xenia, Ohio.

Dr. W. P. Guard (Wash. '36) reports a change of address from Friday Harbor, Wash., to Mount Vernon, same state.

Dr. Phillip P. Poley (Corn. '28), of Kingston, N. Y., recently completed a new home with small-animal hospital attached.

Dr. R. Schaap (Chi. '13), of Pipestone, Minn., has been appointed a member of the Minnesota Veterinary Examining Board.

Dr. Walter J. Williams (Corn. '36), who has been in White Plains, N. Y., until recently, has located permanently at Upperville, Va.

Dr. W. W. Warnock (Ont. '04), of Aledo, Ill., was a patient at the University of Iowa hospital, Iowa City, Iowa, during the early part of June.

Dr. W. N. Cochran (T. H. '12) has resigned from the service of the U. S. Bureau of Animal Industry and has resumed practice at Flat Rock, Ill.

Dr. G. W. Bayles (K. S. C. '37), of Newton, Ill., has located at Marion, Ill., and entered into partnership with Dr. Charles A. Cox (Ind. '14).

Dr. I. S. Alford (Ont. '04), of Paxton, Ill., had several ribs on the right side fractured when he was kicked by a horse he was treating, on June 5.

Dr. R. A. Goodman (Tex. '32), who has been with the U. S. Bureau of Animal Industry in New York City, is now located at Wichita Falls, Texas.

Dr. John H. Lowe (Iowa '00), was retired from the service of the U. S. Bureau of Animal Industry on March 31, 1937. He was stationed at Allentown, Pa.

Dr. Wm. G. Chrisman (Ont. '02), of Chapel Hill, N. Car., was a patient in a Charlottesville, Va., hospital in May, but is reported to be out and around again.

DR. CARL OLSON, JR. (Iowa '31) reports a change of address from Cornell University to Department of Veterinary Science, Massachusetts State College, Amherst, Mass.

DR. C. R. BAUMGARTNER (Chi. '12), of Spencerville, Ind., addressed the Auburn, Ind., Rotary Club on June 14. He urged the production of more live stock in DeKalb County.

DR. JOE F. KNAPPENBERGER (K. S. C. '35), who has been at Kansas State College the past year, has established himself in private practice at 1629 East G Street, Hutchinson, Kan.

DR. JOHN E. LITTLEHALES (McK. '04) has retired from the Royal Canadian Mounted Police after more than 30 years of active service. He is now residing at Esquimalt, B. C.

DR. ORVILLE A. STINGLEY (K. C. V. C. '02) has been retired from active service in the U. S. Bureau of Animal Industry. He was in charge of meat inspection at Topeka, Kan.

DR. EDWIN L. MILLER (Ont. '18), formerly in practice at Newport, Vt., has entered the service of the U. S. Bureau of Animal Industry and is now stationed at Parkersburg, W. Va.

DR. H. H. SEELY (Chi. '10), of Jerseyville, Ill., sustained a fracture of his right forearm when he was kicked by an equine patient, on May 21. Dr. Seely is coroner of Jersey County.

DR. PAUL C. BENNETT (O. S. U. '31) has left the State Laboratories at Reynoldsburg, Ohio, to accept a position in the Veterinary Control Laboratory, University of Wisconsin, Madison, Wis.

DR. SAMUEL BROAD (Ont. '90), of Chesterton, Ind., received internal injuries and a fractured skull when his car was rammed by a truck on U. S. 20, west of Michigan City, Ind., on June 7.

DR. J. H. MURPHY (T. H. '15), of Paris, Ill., addressed the local Rotary Club on June 9. His topic was "The Activities and Accomplishments of the Federal Bureau of Animal Industry."

DR. W. B. EARL (Geo. Wash. '12), of Reno, Nevada, has been appointed a member of the State Board of Veterinary Medical Examiners by Governor Kirman. Dr. Earl succeeds Dr. G. T. Woodward (Wash. '24), of Fallon, who resigned.

DR. WILLIAM R. MICHAEL (Chi. '01), of Highland, Ill., has been made defendant in a damage suit resulting from an automobile accident in which one person was killed and six others were injured. Damages aggregating $55,000 are asked.

DR. ARNOLD S. ROSENWALD (K. S. C. '36) has resigned from the service of the U. S. Bureau of Animal Industry to accept a position as assistant poultry pathologist at the Oregon Agricultural Experiment Station, Corvallis. He assumed his new duties on June 1.

DR. J. W. OLTMANNS (Chi. '16), of Watseka, Ill., talked with his cousin, Dr. J. W. Oltmanns, of Leer, Germany, by radio-telephone, the latter part of May. The two cousins were 4,700 miles apart and the charge for the call was about $36. German coach horses were the subject of the conversation.

DR. B. R. MCCRORY (Colo. '27) has resigned as professor of veterinary medicine at Colorado State College to accept a position with the Black Forest Fox Ranch, at Monument, Colo., as veterinarian and ranch manager. At present the ranch has 750 pairs of foxes, several of which have black and white markings.

DR. E. S. ALLEN (Chi. '15), of Chatham, Va., was seriously injured by a boar while he was attempting to restrain the animal prior to vaccination. The boar inflicted a penetrating wound in one knee, which required hospital attention for several weeks. Latest reports are to the effect that Dr. Allen's condition is favorable.

JOURNAL
of the
American Veterinary Medical Association

Formerly AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

COL. R. J. FOSTER, Pres., Washington, D. C. — M. JACOB, Treas., Knoxville, Tenn.

EXECUTIVE BOARD

CASSIUS WAY, Member-at-Large and Chairman
COL. R. J. FOSTER, ex officio; O. V. BRUMLEY, ex officio
A. E. CAMERON, 1st District; E. P. ALTHOUSE, 2nd District
L. A. MERILLAT, 3rd District; WILLIAM MOORE, 4th District
H. C. H. KERNKAMP, 5th District; I. E. NEWSOM, 6th District
C. H. HAYS, 7th District; L. J. ALLEN, 8th District
H. W. JAKEMAN, 9th District; F. A. ZIMMER, 10th District

The American Veterinary Medical Association is not responsible for views or statements published in the JOURNAL, outside of its own authorized actions. Reprints should be ordered in advance. Prices will be sent upon application.

Vol. XCI, N. S. Vol. 45 — August, 1937 — No. 2

OMAHA IN READINESS

En route, Omaha to Chicago, Aug. 1—Practically the entire day has been spent in conference with the Committee on Local Arrangements, perfecting the details for the program for the 74th annual meeting of the A. V. M. A., which will be held in Omaha during the third week of this month. Veterinarians and their wives, to the number of almost fifty, from points as far east as Ames, Iowa; from Sioux City on the north, and from Lincoln on the west, were in Omaha for the final meetings of their various committees prior to the convention. The chairmen of these groups now report everything in readiness for the two thousand visitors who are expected to attend Omaha's Second A. V. M. A. convention. Probably no meeting of veterinarians has had the publicity that the 1937 A. V. M. A. convention has already received and will receive from now on through the week of August 16.

As for the program, suffice it to say that it would be just about impossible to crowd in another thing. It is solid, from the opening session, on Tuesday, until the close of the clinic, Friday afternoon. Monday undoubtedly will see several hundred

DR. CARL OLSON, JR. (Iowa '31) reports a change of address from Cornell University to Department of Veterinary Science, Massachusetts State College, Amherst, Mass.

DR. C. R. BAUMGARTNER (Chi. '12), of Spencerville, Ind., addressed the Auburn, Ind., Rotary Club on June 14. He urged the production of more live stock in DeKalb County.

DR. JOE F. KNAPPENBERGER (K. S. C. '35), who has been at Kansas State College the past year, has established himself in private practice at 1629 East G Street, Hutchinson, Kan.

DR. JOHN E. LITTLEHALES (McK. '04) has retired from the Royal Canadian Mounted Police after more than 30 years of active service. He is now residing at Esquimalt, B. C.

DR. ORVILLE A. STINGLEY (K. C. V. C. '02) has been retired from active service in the U. S. Bureau of Animal Industry. He was in charge of meat inspection at Topeka, Kan.

DR. EDWIN L. MILLER (Ont. '18), formerly in practice at Newport, Vt., has entered the service of the U. S. Bureau of Animal Industry and is now stationed at Parkersburg, W. Va.

DR. H. H. SEELY (Chi. '10), of Jerseyville, Ill., sustained a fracture of his right forearm when he was kicked by an equine patient, on May 21. Dr. Seely is coroner of Jersey County.

DR. PAUL C. BENNETT (O. S. U. '31) has left the State Laboratories at Reynoldsburg, Ohio, to accept a position in the Veterinary Control Laboratory, University of Wisconsin, Madison, Wis.

DR. SAMUEL BROAD (Ont. '90), of Chesterton, Ind., received internal injuries and a fractured skull when his car was rammed by a truck on U. S. 20, west of Michigan City, Ind., on June 7.

DR. J. H. MURPHY (T. H. '15), of Paris, Ill., addressed the local Rotary Club on June 9. His topic was "The Activities and Accomplishments of the Federal Bureau of Animal Industry."

DR. W. B. EARL (Geo. Wash. '12), of Reno, Nevada, has been appointed a member of the State Board of Veterinary Medical Examiners by Governor Kirman. Dr. Earl succeeds Dr. G. T. Woodward (Wash. '24), of Fallon, who resigned.

DR. WILLIAM R. MICHAEL (Chi. '01), of Highland, Ill., has been made defendant in a damage suit resulting from an automobile accident in which one person was killed and six others were injured. Damages aggregating $55,000 are asked.

DR. ARNOLD S. ROSENWALD (K. S. C. '36) has resigned from the service of the U. S. Bureau of Animal Industry to accept a position as assistant poultry pathologist at the Oregon Agricultural Experiment Station, Corvallis. He assumed his new duties on June 1.

DR. J. W. OLTMANNS (Chi. '16), of Watseka, Ill., talked with his cousin, Dr. J. W. Oltmanns, of Leer, Germany, by radio-telephone, the latter part of May. The two cousins were 4,700 miles apart and the charge for the call was about $36. German coach horses were the subject of the conversation.

DR. B. R. MCCRORY (Colo. '27) has resigned as professor of veterinary medicine at Colorado State College to accept a position with the Black Forest Fox Ranch, at Monument, Colo., as veterinarian and ranch manager. At present the ranch has 750 pairs of foxes, several of which have black and white markings.

DR. E. S. ALLEN (Chi. '15), of Chatham, Va., was seriously injured by a boar while he was attempting to restrain the animal prior to vaccination. The boar inflicted a penetrating wound in one knee, which required hospital attention for several weeks. Latest reports are to the effect that Dr. Allen's condition is favorable.